ROUTINE ANTENATAL SCREENING WITH GLUCOSE CHALLENGE TEST AND ITS RELATION WITH PERINATAL OUTCOME

DISSERTATION SUBMITTED IN FULFILLMENT OF THE

REGULATIONS FOR THE AWARD OF

M.D.OBSTETRICS AND GYNAECOLOGY

DIVISION OF OBSTETRICS AND GYNAECOLOGY

PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

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GUIDE

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INTRODUCTION

Pregnancy is time when serial metabolic changes in mother are carefully regulated so as to provide optimum substrate to both mother and foetus. Subtle perturbation in maternal metabolism can have implication not only for index pregnancy but also for the future generation.

Diabetes is one of the commonest medical complication of pregnancy. Patients can be separated into those who were known to have diabetes before pregnancy (overt) and those diagnosed during pregnancy (gestational).

GDM is defined as carbohydrate intolerance of varying severity with onset or first recognition during pregnancy.

GDM as a concept began in 1964 when O' Sullivan and Mahan performed a 100gm 3 hour GTT on 752 pregnant women with atleast two values above two standard deviation.

GDM is risk factor for the mother and the foetus. The risk increase proportionally to the maternal blood sugar concentration along a glycaemic continuum. Various screening and diagnostic tests are used. The need to screen the GDM as a risk factor in the whole population of pregnant woman has lead us to propose the use of simple, universally applied test constituted by glucose challenge test.

SCREENING FOR ABNORMAL GLUCOSE TOLERANCE

IN PREGNANCY

The purpose of screening is to subject minimum number of women to the diagnostic test – the oral Glucose Tolerance Test (high specificity) and yet to detect as many as possible cases (high sensitivity). Ideally screening should be performed at the initial visit in order to detect the rare, previously undiagnosed cases of subclinical diabetes mellitus. This is particularly justifiable in some groups (ie) Impaired Glucose Tolerance / Gestational Diabetes Mellitus in a previous pregnancy or in high risk women such as older Indian women with multiple potential diabetic features, screening test should be repeated again even if an earlier test is negative. As a compromise 28 weeks is usually considered an appropriate time as most cases should be diagnosed by then and there may be an opportunity still to influence the outcome.

Screening tests are means by which patients are selected for definitive testing. screening tests should be well defined, clearly administered, inexpensive, reproducible and have high sensitivity. It need not have high specificity demanded of the diagnostic test.

Tests of carbohydrate intolerance during pregnancy

- Simple screening test, abnormal value include fasting plasma glucose >105 mg% and random 2 hours / post prandial glucose 120 mg%
- Recommended loading test for screening, 50 gm glucose load given orally at random, abnormal is >140 mg% after 1 hour.
- Other screening tests for abnormal glucose tolerance

GLUCOSURIA AS A SCREENING TEST

Commonly employed screening test for detection of glucose intolerance. But during pregnancy renal threshold for glucose is often lowered partly due to an 8 fold increase in

glomerular filtration of glucose and partly to an intermittent tubular defect in glucose reabsorption.

The specificity can be increased by defining significant glycosuria as that which occurs in second fasting specimen. The patient is instructed to void on waking up. Half an hour later while still fasting, she voids again and the specimen is tested for glucose. In normal pregnancy, fasting blood glucose is low so glucose at that time cannot be due to low renal threshold and is thus significant. Women with renal glycosuria during pregnancy are at a high risk of premature delivery (25% incidence) and the development of fetal macrosomia (7%) in some cases renal glycosuria during pregnancy is a manifestation of renal tubular damage caused by chronic nephritis.

STOVER (1982) found glucosuria in fasting specimen to be a true indicator of glucose intolerance during pregnancy.

FASTING GLUCOSE STUDIES

In fasting states there is accelerated starvation in the mother. After an overnight fasting of 12 hour, the level of glucose in pregnancy is significantly lower compared to non pregnant state (ie) an exaggerated lowering of circulating glucose concentration.

METZGEN et al reported that plasma glucose concentration after an overnight fasting was approximately 10 mg% lower in pregnant women and that glucose fall by additional 8 – 10 mg% in the pregnant but not in non-pregnant, when both groups postponed breakfast for 6 hours. When there is fasting plasma glucose value >105 mg%, it suggests glucose intolerance.

O'SULLIVANS GLUCOSE CHALLENGE TEST

O' Sullivan's 50 gm oral glucose challenge test is the best screening test with excellent sensitivity and specificity of 79% and 87% respectively (GABBE et al 1991). The 1990 Chicago

conference on gestational diabetes recommended that all pregnant women should be screened using a 50gm oral glucose challenge test between 24 to 28 weeks of gestation without regard to the time of the day or last meal and that a plasma value at 1 hour exceeding 140 mg/dl be used as the cutoff for performing the diagnostic 100gm oral glucose tolerance test. Women with previous history of GDM may benefit from earlier screening. If screening in early pregnancy yields a normal result, subsequent screening should be performed at 24 to 28 weeks.

According to ACOG (1994) the sensitivity of screening may be improved by using a 130 mg/dl threshold rather than 140 mg /dl to define an abnormal response to 50 gm test. Use of lower threshold value may increase detection of abnormal glucose tolerance from 90% to nearly 100% but at the expense of subjecting 25% of pregnant women to the 3 hour test. Thus the threshold for further testing be chosen based on goal of screening.

GLUCOSE POLYMER CHALLENGE TEST

Glucose polymer is an inexpensive commercially available glucose saccharine mixture containing 3% glucose, 7% maltose, 55% maltotriose and 85% polysaccharides. Its osmotic load is one fifth that of glucose and has been associated with gastrointestinal symptoms. A moderate level of agreement between the results of 3 hour GTT has also been demonstrated.

GLYCATED BLOOD PROTEINS IN THE DIAGNOSIS OF GDM

Glycated Hb% and other proteins have been investigated as screening test for GDM. Glycation is the slow and almost irreversible binding of glucose or a phosphorylated sugar to Hb or other blood proteins. Because it is dependent on concentration of reactants and because the red cell concentration of glucose approximates that in extracellular fluid glycated Hb% has been investigated as a diagnostic test for non gestational related diabetes.

GDM however may not present with the same constant elevation of blood sugar levels as in non pregnant state. Gravid women with GDM have fasting blood sugar level that is low. Because of increased erythropoisis, red blood cells are younger in pregnancy, Hb is less glycated. Hormonal milieu changing rapidly from relative insulin sensitivity to that of insulin resistance as pregnancy progresses, a measure of chronic hyperglycemia such as glycated Hb% may not be effective in GDM.

SHAH et al measured Hb A1C using ion exchange chromatography and applied NDDG criteria for GDM in a group of patients. The results showed 27% sensitivity for glycated Hb% in the identification of GDM in risk group. Hb A1C >8.8% taken as abnormal. These data do not support the use of Hb A1C as screening test for GDM.

Other glycosylated plasma proteins that are used as potential marker are glycosylated albumin and fructosamine. It is commonly used in clinical investigation because it models the physiological events after a meal and is easily administered. The national diabetes data group (NDDG) criteria for diagnosis of GDM has been recommended by American diabetes association.

	O'Sullivan (whole blood)	NDDG (P)	Carpenter Coustan(P)
Fasting	**90 (5)**	**105 (5.83)**	**95 (5.28)**
I hr.	**165 (9.17)**	**190 (10.56)**	**180 (10)**

2 hr.	**145 (8.06)**	**165 (9.17)**	**155 (8.61)**
3 hr.	**125 (6.94)**	**145 (8.06)**	**140 (7.78)**

If more than or equal to 2 values are met or exceeded – diagnosis of GDM is made.

DETERMINANTS OF ABNORMAL GLUCOSE TOLERANCE IN PREGNANCY

The various risk factors associated with abnormal glucose tolerance in pregnancy are :

- Age
- Ethnicity
- Obesity (>200pounds or 15% of non pregnant ideal body weight)
- Positive family history of diabetes
- Poor reproductive history
  (>3 spontaneous abortions in the first or second trimester)
- History of prematurity
- History of stillbirth
- History of delivery of large infant
- History of unexplained neonatal death
- History of traumatic delivery with associated neurological disorder in the infant
- History of congenital anomaly
- History of diabetes in previous pregnancy
- History of preeclampsia in a previous pregnancy, chronic hypertension
- Preeclampsia in a multipara
- Recurrent severe moniliasis or UTI
- Polyhydramnios

- Glycosuria

ETHNICITY

The prevalence of abnormal glucose tolerance is highly dependent on ethnicity (HADDEN 1985 BEISCHER et al 1991). Compared with European women, the prevalence rate is approximately eleven fold in women from Indian sub continent (DORN HORST et al 1992)

AGE AND OBESITY

Increasing age and obesity (BMI>26) are independent risk factors for abnormal glucose tolerance (O'SULLIVAN et al 1973; MARESH & BEARD 1989; ROSEMAN et al 1991)

FAMILY HISTORY OF DIABETES

Family history of diabetes either in first degree or second degree relatives is an important risk factor for developing abnormal glucose tolerance (5 –10 times risk greater than for children with non diabetic parents).

The risk for IDDM is found to be inherited asymmetrically from the mother and father.

GARNER et al (1995) observed when the mother had IDDM, 1-3% of offsprings developed diabetes whereas when the father had IDDM, 6% of offsprings developed diabetes and when both parents had IDDM, 20% of offspring developed diabetes. The greatest risk of the offspring developing diabetes occurs when one or both parents developed the disease before the age of 40.

Hence a history of IDDM in the father is of greater predictive value than in the mother and even greater when a sibling is diabetic. A family history in grandparents is less significant.

FOSTER (1994) found that 40% of siblings and one third of offsprings of women with NIDDM develop abnormal glucose tolerance or obvious diabetes.

FETAL & PERINATAL WASTAGE

Pregnancy outcome is highly dependent on maternal glycemic control throughout the pregnancy. Several studies have shown that spontaneous abortion is associated with poor glycaemic control during the first trimester, GREENE et al (1989), MILLS et al (1988b). KARLSON & KJELLMER 1972 demonstrated an inverse relationship between mean ambient glucose levels during third trimester and perinatal mortality. HANSON & PERRSONN (1993) in

the Swedish study reported the incidence of unexplained stillbirths to be between 0.4 to 3% and the incidence of preterm labour in classes B to F diabetes as 25%.

PERINATAL MORBIDITY AND MORTALITY

This is increased due to the occurence of following neonatal complications.

(a) Neonatal hypoglycaemia:

Defined by the ACOG (1995) as plasma glucose < 35 mg/dl (1.7mmol/l) in a term fetus. It occurs due to hyperinsulinaemia and suppression of endogenous glucose production by decreasing gluconeogenesis and glycogenolysis which occurs despite an abundance of glycogen stores in liver and myocardium. Hyperinsulinaemia also leads to increased peripheral glucose utilisation. The peak age of onset of hypoglycaemia is at 1 to 1.5 hours of age. The factors mainly protective against foetal hypoglycaemia is the optimal control of maternal hyperglycaemia especially during the third trimester and during labour. It has been shown that a mean maternal plasma glucose >105 mg/dl during the last 4 hrs. of labour in a diabetic mother leads to higher incidence of neonatal hypoglycaemia.

(b) Hypocalcaemia :

About 25% of the infants of diabetic mothers may present with serum calcium <7mg/dl (1.6mmol/l). The mechanism remains under investigation, but it has been suggested that a state of relative maternal hyperparathyroidism play a role. Asphyxia and prematurity operating through elevated cortisol induces vitamin - D antagonism at the intestinal level. Respiratory distress and fetal metabolic acidosis may result in calcium being shifted from intracellular to extra cellular pools and reversal of this shift during the correction of acidotic event may produce hypocalcemia. The peak age of onset hypocalcaemia is the second or third day of life. Hypomagnesemia may coexist.

(c) Birth injury :

Some of the birth injuries in macrosomic infants include Erb's palsy, fractured clavicle, facial paralysis, phrenic nerve injury, intracranial haemorrhage in the form of intra cerebral bleeding or subdural hematoma.

(d) Respiratory distress syndrome :

RDS has been observed in about 5% of the infants. In vitro studies indicate that insulin antagonises the stimulatory effects of cortisol on fibroblast to induce the synthesis of fibroblast – pneumocyte factor (FPF) which in turn inhibits type II cells and phosphatidyl choline production. But several reports have challenged the concept of diabetes altered lung function. Gestational age, rather than overt diabetes, is likely the most significant factor governing the development of RDS (BERKOWITZ et al 1996).

(e) Neonatal hyperbilirubinaemia :

The pathogenesis of hyperbilirubinemia is uncertain. Factors implicated are prematurity and polycythemia with hemolysis.

(f) Neonatal polycythemia and hyperviscosity syndrome :

This is diagnosed when the neonatal hematocrit exceeds 65% and has been observed in as many as 40% of infants of diabetic mothers (SALVESEN et al 1992). It occurs probably due to excessive production of erythropoietin. Diabetic pregnancy may also contribute. The resultant hyperviscosity may induce congestive heart failure and vascular thrombosis accounting for the increased risk of renal vein thrombosis in these infants.

(g) Fetal cardiac hypertrophy :

Fetal hyperinsulinaemia may lead to hypertrophic cardiomyopathy that occasionally progresses to congestive cardiac failure

(GANDHI et al 1995).

(h) Long term sequelae:

a. Growth – The growth rate, height and development as they relate to obesity are excessive during childhood and early adulthood which suggests an important influence of the diabetic intrauterine environment.

- Glucose hemostasis – Because of genetic and environmental factors, glucose tolerance is more likely to be abnormal in these offsprings. One third of offsprings of NIDDM develop abnormal glucose tolerance or obvious diabetes (FOSTER 1994).

- Neurologic and psychological development : Offspring of high risk pregnancy often have neurologic deficits which are relatively minor but which may be a significant cause of morbidity. Possible reasons for these are birth trauma, metabolic abnormalities during and after gestation, cerebral dysfunction etc.

CONGENITAL MALFORMATIONS

HENRIQUES et al (1991) found that diabetes is not associated with increased risk for fetal chromosomal abnormalities. ROSSEN et al 1994 demonstrated the incidence of major malformations in women with overt diabetes is 5 – 10%.

FETAL MACROSOMIA

WILLIAM et al 1986 have shown that the incidence of macrosomia rises significantly when mean maternal blood glucose concentrations exceed 130 mg/dl. PETTIT et al found a direct relationship between the maternal plasma glucose 2 hours after a 75 gm oral glucose challenge and the likelihood of the birth of a large baby.

PREVIOUS HISTORY OF ABNORMAL GLUCOSE TOLERANCE

Women with history of abnormal glucose tolerance are at greatly increased risk of diabetes in future pregnancies (PHILIPSON & SUPER 1989, CARPENTER et al 1996 and CURET et al 1996) observed that women diagnosed to have abnormal glucose tolerance have pre-existing alterations in insulin economy and BERKUS et al 1996 concluded that gestational diabetes was likely to be maturity onset diabetes rather than a distinct diabetic state due to pregnancy.

HYPERTENSION

SUHONEN and TERANO 1993 studied hypertension and pre-eclampsia in women with gestational glucose intolerance and found an increased incidence in these patients. SOLOMON et al 1994 have found that glucose intolerance was an important predictor of hypertension in pregnancy.

INFECTIONS

STAMLER et al 1990 found an increased rate of infections morbidity in antenatal patients with abnormal glucose tolerance. 80% had candida vaginitis, pyelonephritis, skin and respiratory infections and pelvic puerperal infections.

REVIEW OF LITERATURE

O'SULLIVAN and MAHAN in their classical study of 1964 analysed glucose response over 3 hours to a 100 gm oral glucose challenge in 752 healthy pregnant women which yields values representing the mean plus or minus two standard deviation in the fasting state and at 1,2 & 3 hours by arbitrarily declaring abnormal carbohydrate handling as that exceeding 2.5 D above the mean on 2 or more values, 2.5% of the population was defined as having GDM.

Diagnosing GDM and instituting aggressive management of the mother are intended to reduce or eliminate the perinatal, neonatal and long term complication in the offspring. O'Sullivan and Mahan's criteria was too lax for the identification of people at risk for perinatal morbidity associated with carbohydrate intolerance. The original criteria was based on whole-blood determination of glucose by the Somogyi method.

This was subsequently modified by National Diabetes Data Group to use a conversion factor of 1.14 represent plasma glucose determination by glucokinase technique. Technical modification of that conversion have been recommended by Carpenter and Coustan as being more representative of the true plasma glucose determination. This modification results in a lowering of all glucose levels in the 3 hours glucose tolerance test, thus increasing the sensitivity of the test. By using the lower modified criteria, overall incidence of GDM is increased by 56%. Data on the modified criteria presented at the 4^{th} international workshop on gestational diabetes infants of women meeting these lower criteria are at a risk for perinatal morbidity including macrosomia. Hence, Carpenter and Coustan criteria was adopted for diagnosis.

The 4^{th} international workshop on gestational diabetes defined cut off values for the controversial 75g oral GTT in pregnancy. The cut off values were arbitrarily defined

based on the mean plus 1.5 SD of the OGTT values in a study of over 3500 patients. Greater experience in the use of 75gm oral GTT and maternal and infant outcomes data will be needed to define better cut off values for this test. Data are becoming increasingly available to suggest that a single abnormal value on GTT may predict perinatal outcome. Tallerigo et al examined the neonatal outcome in 249 women and found that 2 hours plasma glucose concentration after a 100gm OGTT significantly correlated with the infant's birth weight.

Because of lack of reproducibility of the glucose tolerance test, together with the discrepancies in the number of abnormalities much effort has gone into establishing simpler diagnostic criteria for GDM. Neither glycated haemoglobin nor fructosamine is sufficiently sensitive for identification of women with GDM.

Random glucose testing and use of reflectancemeters lack the sensitivity for adequate identification of women at risk for GDM. The best screening test appears to be the 50gm, 1hour glucose challenge test. The second international workshop conference on GDM concluded that all pregnant women should be screened for GDM. A 1 hour plasma glucose determination in excess of 140gm/dl (lower by Carpenter Coustan criteria) constitutes a positive screen and requires the performance of a traditional 100gm OGTT for confirmation of GDM.

Nayeor et al evaluated data on over 3000 pregnant women and developed a scoring system to determine the risk of GDM based on age, BMI and race. The American diabetes association position statement suggest that it is not cost effective to screen women at low risk. This new policy has been controversial, however, with some suggesting that 10% of patients with GDM would be missed if all women were not screened.

Glucose traditionally has been used as the marker for GDM because of its ease of measurement and test reproducibility. It is now clear that alteration in insulin secretion, insulin sensitivity and carbohydrate, fat and amino acid metabolism are all intrinsic abnormalities in the state that we have to accept as GDM. Developing more sensitive indices for prediction of perinatal morbidity may require either intensification of glycaemic criteria or the inclusion of more sophisticated metabolic measurements.

The second international workshop on gestational diabetes in 1985 defined gestational diabetes as "carbohydrate intolerance of varying severity with onset or first recognition during present pregnancy" and this is the current widely accepted definition of GDM.

Gabbe in 1980 in his masterly review titled "management of diabetes in pregnancy ; six decades of experience" traced the history of management of this condition and identified four distinct periods as shown below :

AIM OF CARE

- 1921 – 1940 Avoid ketoacidosis
- 1941 – 1970 Team care / Early delivery
- 1971 – 1976 Foetal Surveillance
- 1976 – 1992 Aim for normoglycemia

DAVID STAMILIO et al in January 2004 performed a retrospective cohort study of 1825 eligible pregnant women among a cohort of 1998 patients. Patients were screened for GDM with the 1 hour 50gm GCT at 24 - 28 gestational week. False positive GCT was defined as a result greater than or equal to 135 mg./ dl followed by a normal 3 hour GTT. Comparison was made between negative GCT and false positive GCT for a composite perinatal outcome variable that included foetal macrosomia, antenatal death, shoulder

dystocia, chorioamnionitis, preeclampsia, NICU admission, caesarean delivery and postpartum endometritis. The results were 164 patients with a false positive GCT and 50 patients with GDM. The false positive GCT cohort on average was older, was of a higher parity, had a higher BMI and more frequently had chronic hypertension, sickle cell trait and elevated midtrimester HCG. False positive GCT was more frequently associated with adverse perinatal outcome including composite perinatal out come (Odds ratio 5.96), macrosomia more than 4500 g (OR 3.66), antenatal death (OR 4.61), shoulder dystocia (OR 2.85), endometritis (OR 2.18), and caesarean delivery (OR 1.76).

The university of Pennsylvania institutional review board approved this study. So patient with a false positive GCT could benefit from additional therapies such as more intensive foetal monitoring, nutritional counselling or a diabetic diet. The results of this study suggests that having a false positive GCT is an independent risk factor for adverse perinatal outcome.

Rey et al reported that patients with an abnormal GCT and a single elevated value on the GTT are at increased risk for foetal macrosomia, neonatal hypoglycemia and neonatal hyperbilirubinaemia.

Okun et al showed that patients with an abnormal GCT and no elevated values on the GTT are at increased risk for foetal macrosomia.

Sun et al, in 1995 did a prospective study on the relationship between 50gm. GCT and pregnancy outcome. 50gm OGCT was performed on 622 pregnant women and 75gm OGTT was further done on subjects with screening test value of more or equal to 7.78mmol/l. 16.56% (103/622) had increased GCT. Among whom 32 were identified as having GIGT and 12 , GDM by confirmatory test of 75gm GTT. Sensitivity was 42.72% (44/103). The incidences of EPH syndrome, PROM, foetal macrosomia, operative deliveries and perinatal morbidity were higher in women with GIGT/GDM than in women without

GIGT/GDM. It suggests that 50gm GCT is ideal method of screening for GDM and should be performed on all pregnant women.

Bevier et al studied 103 women who had positive GCT (>140 mg/dl) but a negative 100 gm 3 hour OGTT. The women were randomly assigned to either experimental or control groups with experimental receiving dietary counselling and home glucose monitoring instruction (HBGM). They were reviewed weekly. All women had HbA_1C tests at 28 and 32 weeks. Results were all women whom were in dietary counselling had low infant birth weight, less number of caesarean sections and low HbA_1C.

Bonomo et al in 1998 suggested maintaining the 140 mg/dl oral GCT threshold with diagnostic target is to recognise only women with positive results of oral GTT. To prevent perinatal risks in pregnancies complicated by borderline glucose intolerance, with Carpenter Coustan criteria lower cut-off value (136mg/dl), could be hypothesised to improve test sensitivity, allowing more extensive diagnosis of borderline subjects.

Institute of obstetrics and gynaecology, university of Florence, Italy in 1997 studied whether minor abnormalities of glucose metabolism without gestational diabetes are risk factors for foetal over growth. A sample of 1883 unselected pregnant women were screened for GDM using 50gm of glucose, 1 hour GCT in 2 periods of pregnancy, early (16 – 20weeks) and late (26 - 30 weeks). Results were the level of risk was related to gestational age at the appearance of abnormal GCT. Patients with an abnormal GCT in the early and late periods of pregnancy had a risk of delivering a large for GA infant 7 times higher than the control group (normal GCT in both periods) and patients with a abnormal GCT in the early period and abnormal GCT in the late period showed a risk 3 times higher than the control group.

Lao et al in 2002 in Queen Mary Hospital, Hongkong studied on 461 LGA babies, the relationship between WHO category of IGT (2 hr value of 75gm OGTT at 8 to

10.9mmol/l) and outcome in LGA infants to determine whether IGT affects perinatal morbidity in addition to affecting infant size. IGT group had significantly higher mean maternal age, pre-pregnancy weight and BMI, but no difference in infant gestational age and birth weight. However, IGT group had increased incidence of erb's palsy, meconium aspiration syndrome, phototherapy, sepsis and shoulder dystocia.

AIM OF STUDY

- To do routine antenatal screening with glucose challenge test and to study the incidence of GDM in the study group and positive predictive value of GCT in pregnant women attending antenatal OP of PSGIMS&R during a period of July 2004 – July 2005.

- To study the incidence of GCT positive GTT negative patients (false positive GCT).

- To study the validity of GCT in diagnosing GDM.

- To study the maternal and perinatal outcomes in both GCT positive GTT negative patients and GDM patients.

- To compare perinatal outcomes between patients with GCT+ GTT- patients and patients with normal GCT.

MATERIALS AND METHODS

A prospective study was carried out in pregnant women attending antenatal op in PSGIMS&R and all of them were followed till delivery to assess the maternal and perinatal outcomes.

SELECTION CRITERIA

Pregnant women attending antenatal OPD were recruited for the study

INCLUSION CRITERIA

- All pregnant women with gestational age between 24 – 28 weeks
- Any parity
- Any age
- Not a known DM

EXCLUSION CRITERIA

- Gestational age of more than 28 weeks
- Known DM
- Multiple pregnancy
- Other medical and surgical disorders complicating pregnancy like cushings disease, pancreatitis, hyperthyroidism etc
- On drugs that would alter glucose and insulin metabolism like corticosteroids, thiazides, salbutamol, nifedepine etc

METHODS

A detailed history including the various risk factors were taken :

- Age
- Ethnicity
- Obesity

- Positive family history of DM
- Poor reproductive history
- Previous h/o prematurity and stillbirth
- H/o delivery of large infant
- H/o unexplained neonatal death
- H/o traumatic delivery with associated neurological disorder in the infant
- H/o congenital anomaly
- H/o DM in the previous pregnancy
- H/o preeclampsia in previous pregnancy
- Recurrent severe moniliasis or UTI
- Polyhydramnios
- Glycosuria

O' Sullivans glucose challenge test

50 gms of oral glucose mixed with 150 ml of water was given to all patients without regard to the time of the last meal or time of the day. Glucose given was Dextrose Monohydrate D Glucose which is commercially available. Following the glucose drink, patients were made to rest and prohibited from further eating or drinking except water. One hour later 1cc of venous blood was taken in a dry test tube and plasma glucose was estimated by enzymatic method (glucose oxidase method). Inorder to achieve increased sensitivity, according to ACOG 1994 recommendations, a cut-off value of 130 can be taken. But conventionally >140 mgs% is considered as positive screening.

3 Hour Oral Glucose Tolerance Test

Patients whose GCT were above 140 mgs% were subjected to 3 hr oral GTT, Since it served as gold standard for diagnosis of GDM. After taking the fasting sample (blood and urine), patients were challenged with 100 gms of glucose. Venous blood was taken

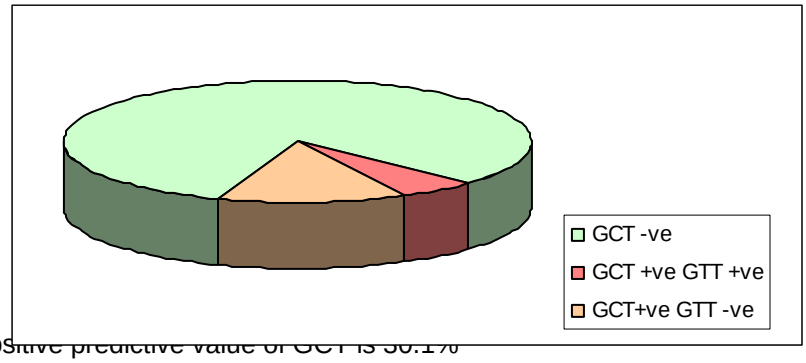
hourly for a period of 3hrs along with collection of urine specimen. The diagnostic criteria followed by NDDG is

Fasting	1 hr	2hr	3hr
105	190	165	145mg/dl
5.8	10.6	9.2	8.1mmol/l

If any two values were met or exceeded, a diagnosis of gestational diabetes was made and if the 2 hour plasma glucose was between 115 and 164 mg%, a diagnosis of IGT was made and they were managed accordingly. All patients were followed up till delivery and the perinatal outcome was analysed.

RESULTS AND ANALYSIS

- Total no of antenatal patients screened with GCT – 500
- No of patients with positive GCT (>140mg/dl) – 93
- No of GCT + patients in whom GTT is positive _ 28
- Therefore, no. of patients with GCT +GTT-VE – 65
- No of patients with negative GCT (>140mg/dl) – 407



⇨ Positive predictive value of GCT is 50.1%

⇨ Incidence of GDM is 5.6%

⇨ Incidence of GCT+GTT-VE patients is 13.77%

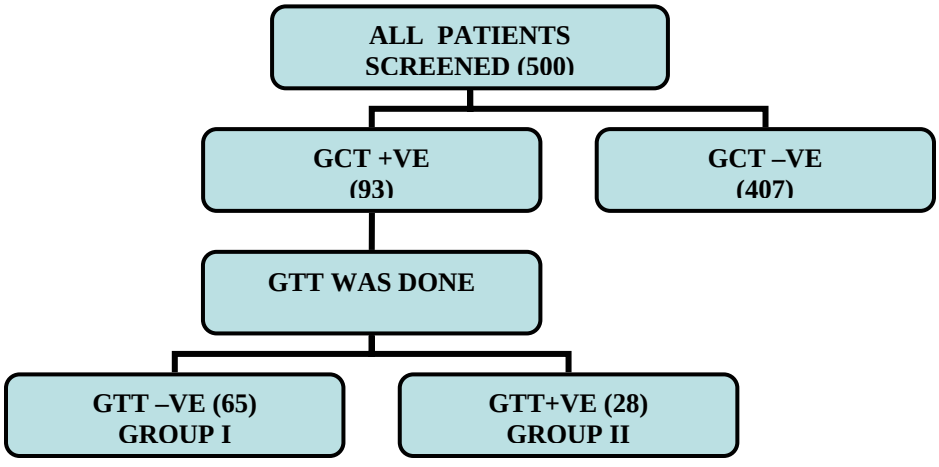


TABLE – 1

AGE GROUP

AGE	NO.OF PTS.(500)	GCT+GTT-VE (65)		GDM (28)	
		No	%	No	%
<20	34	2	5.9	_	_
21-30	407	47	11.5	24	5.9
>31	59	16	27.1	4	6.8

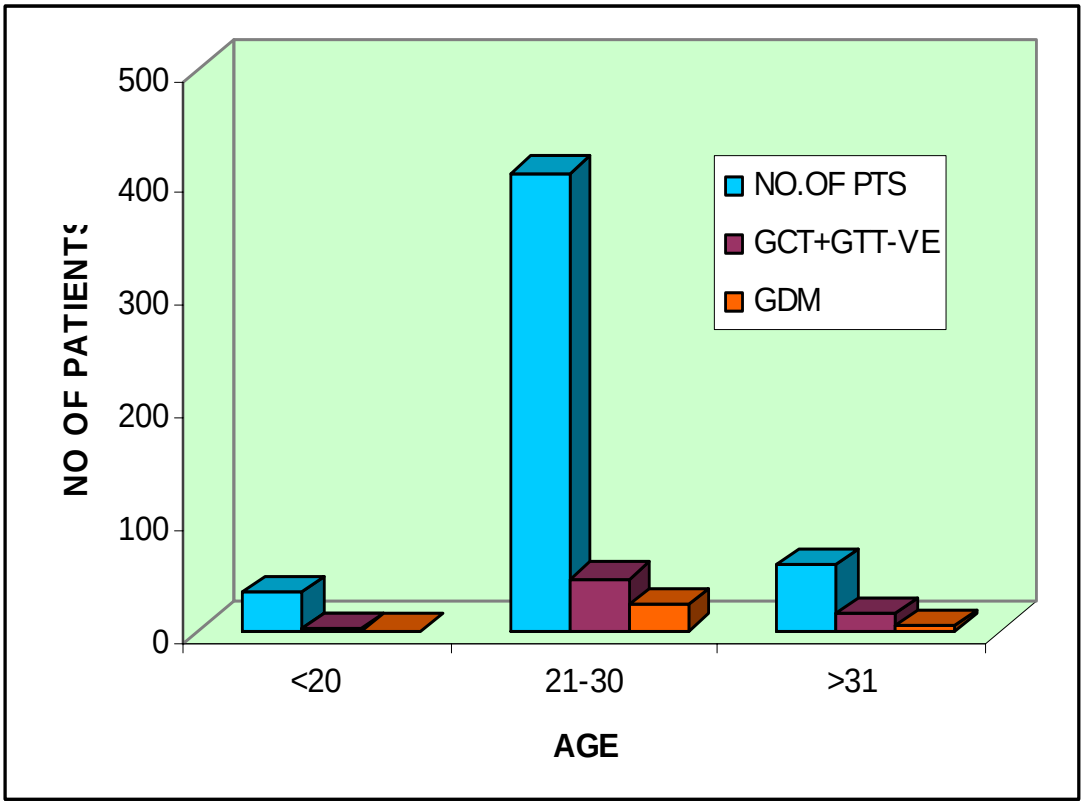


TABLE-2

GRAVIDITY

Gravida	No of pts (500)	GCT+GTT-(65)		GDM (28)	
		No	%	No	%
1	227	23	10.1	10	4.4
2	178	22	12.4	12	6.7
3	68	19	27.9	3	4.4
4	19	1	5.3	3	15.8
5	8	_	_	_	_

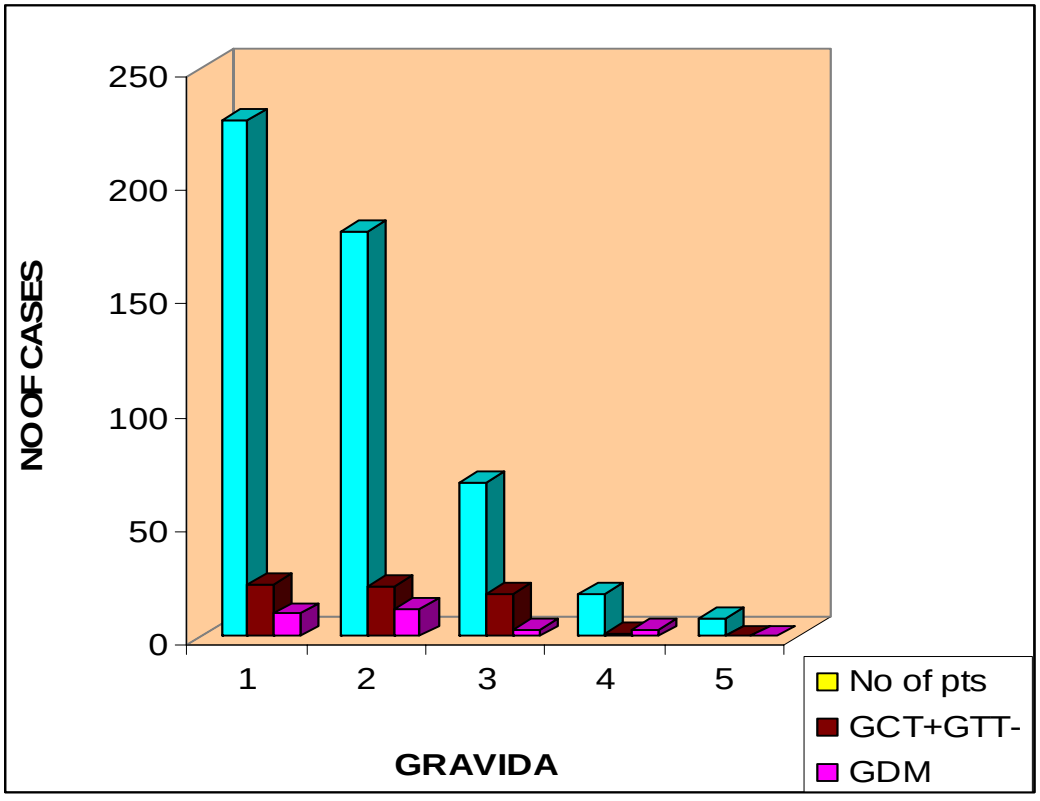


TABLE -3

SOCIO ECONOMIC STATUS

SES	NO OF PTS (500)	GCT+GTT-VE(65)		GDM(28)	
		No	%	N0	%
I	_	_	_	_	_
II	18	3	16.7	2	11.1
III	71	24	33.8	11	15.5
IV	353	30	8.5	13	3.9
V	58	8	13.8	2	3.4

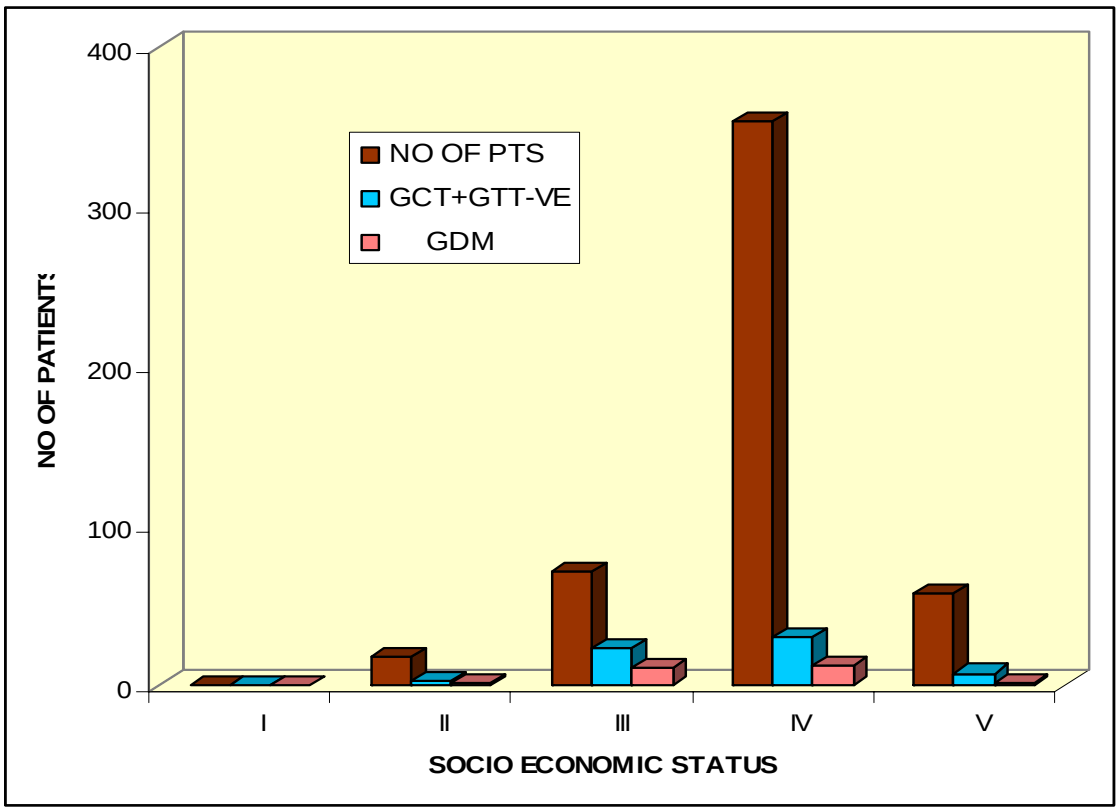


TABLE -4

BMI

BMI	NO OF PTS (500)	GCT+GTT-VE (65)		GDM (28)	
		No	%	No	%
<20	6	1	16.7	_	_
21 – 25	181	19	10.5	5	2.8
26 -30	244	33	13.5	12	4.9
>31	69	12	17.4	11	15.9

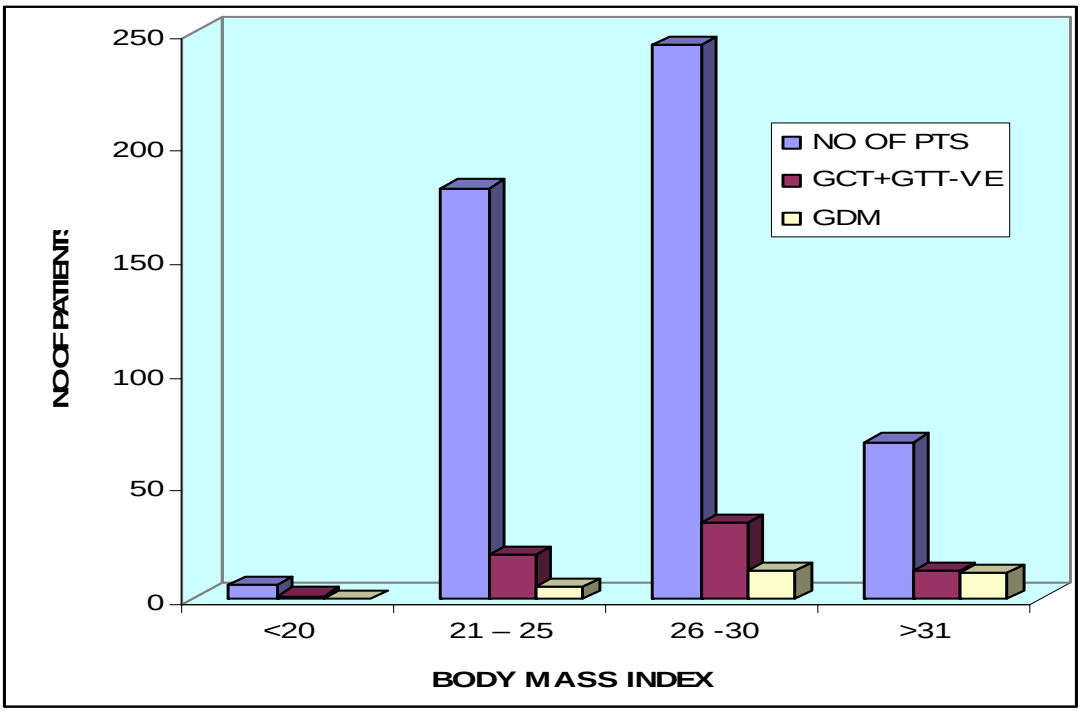


TABLE – 5

FAMILY H/O DIABETES

FAMILY H/O DM	NO OF PTS (500)	GCT+GTT-VE(65)		GDM (28)	
		No	%	No	%
FATHER	28	9	32.1	4	14.3
MOTHER	18	4	22.2	4	22.2
BOTH	3	1	33.3	1	33.3

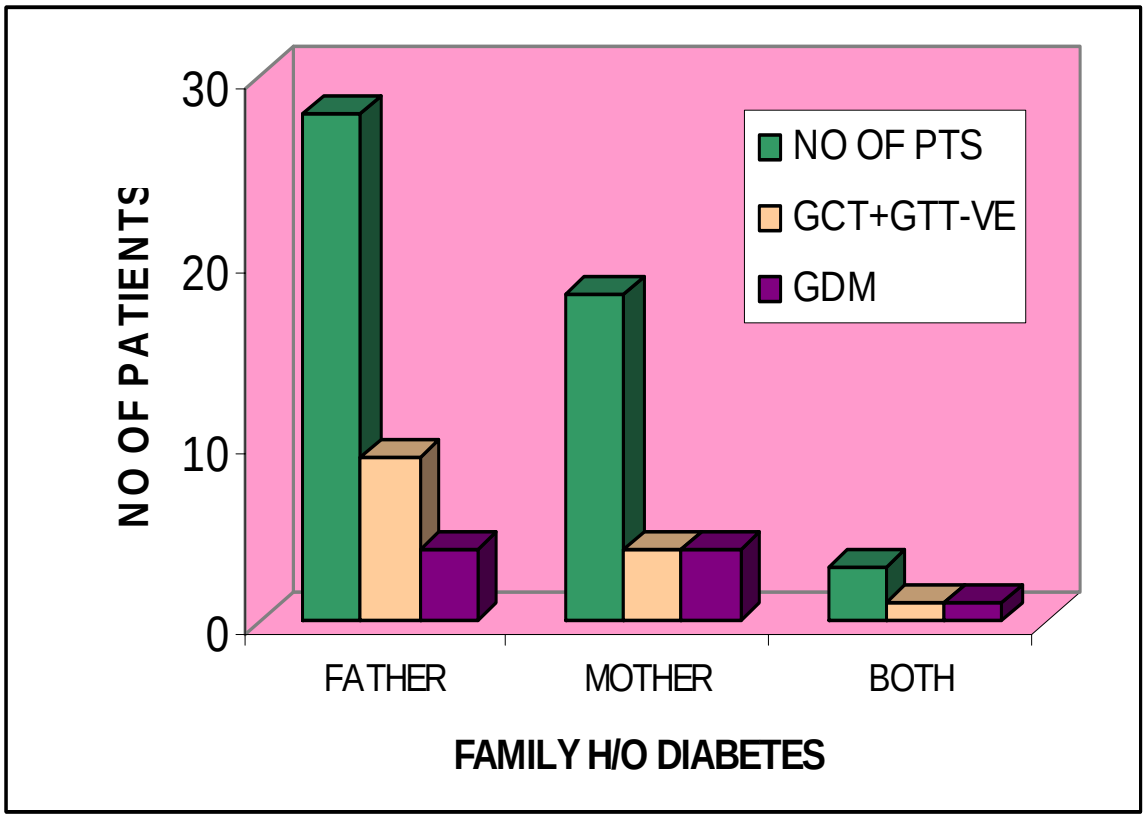


TABLE – 6

POSITIVE URINE SUGAR

URINE SUGAR	TOTAL NO OF PTS(500)	GCT+GTT- (65)		GDM (28)	
		No	%	No	%
POSITIVE	7	2	28.6	3	42.9

PRESENT OBSTETRIC HISTORY

Total no of AN women screened – 500

PREECLAMPSIA

- ⇨ Total no of women with PIH -36

  (mild – 33;severe -3)

- ⇨ NO of women with group I with PIH – 8

  (mild -7;severe -1)

PRESENT OBSTETRIC HISTORY	TOTAL NO OF PATIENTS(500)	GCT+GTT-VE(65)		GDM(28)	
		No	%	NO	%
PREECLAMPSIA	36	8	12.3	2	7.1

HYDRAMNIOS

PRESENT OBSTETRIC HISTORY	TOTAL NO OF PATIENTS(500)	GCT+GTT-VE(65)		GDM(28)	
		No	%	NO	%
HYDRAMNIOS	7	2	3.1	2	7.1

PRETERM DELIVERY

PRESENT OBSTETRIC HISTORY	TOTAL NO OF PATIENTS(500)	GCT+GTT-VE(65)		GDM(28)	
		No	%	NO	%
PRETERM LABOUR	28	2	3.1	1	3.6

H/O PREVIOUS LSCS

- Total no of cases – 59
- GCT +GTT- cases with previous lscs - 8
- GDM with previous lscs – 3

PREVIOUS OBSTETRIC HISTORY

In GCT + GCT –VE :

- Previous 2 neonatal deaths (1 due to severe PIH)
- Previous anencephaly – 2
- Previous hydrocephalus - 1
- Anomaly – 1 (diaphraghmatic hernia)

In GDM :

- Previous neonatal deaths – 2
- Previous IUD – 2 (1 was due to severe PIH)
- H/O GDM in previous pregnancy – 1
- H/O previous macrosomia – 1 (4.2 kgs)

TABLE - 7

GESTATIONAL AGE

GESTATIONAL AGE	TOTAL NO OF PTS(500)	GCT+GTT-(65)		GDM (28)	
		No	%	NO	%
TERM	472	63	13.3	27	5.7
PRETERM	28	2	7.1	1	3.6

TABLE -8

MANAGEMENT

TREATMENT	NO OF GDM	%
MEALPLAN	20	71.4
INSULIN	8	28.6

TABLE – 9

ONSET OF LABOUR

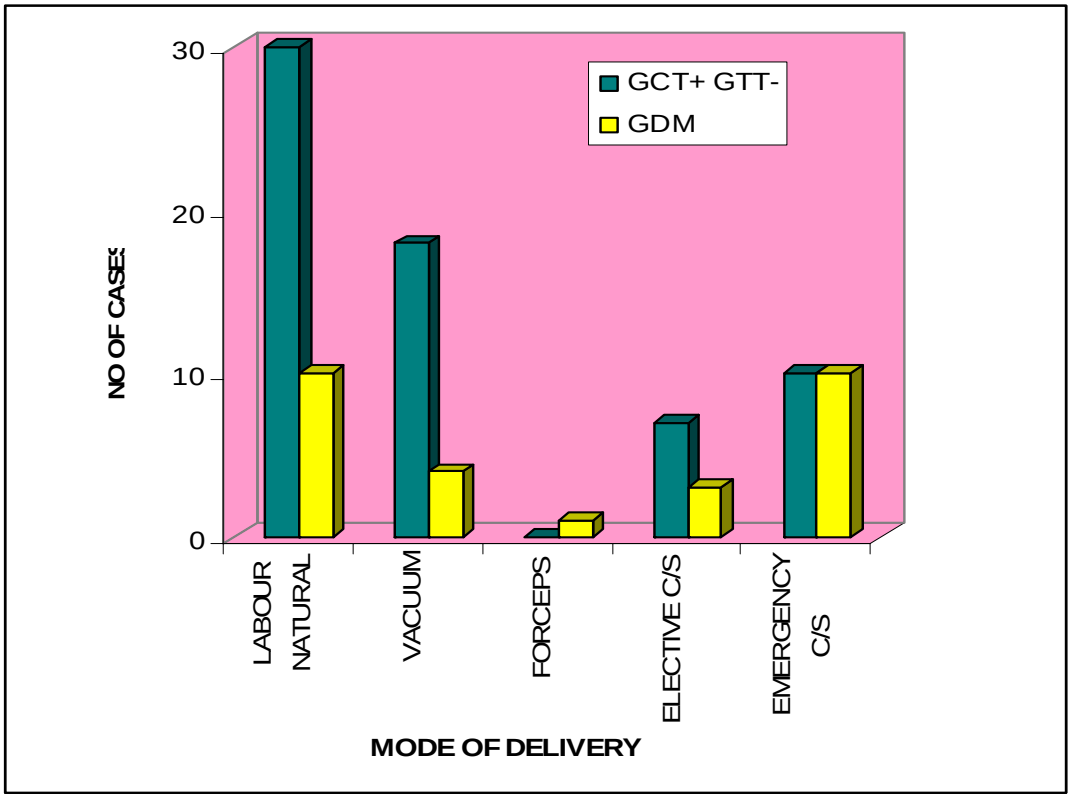
ONSET OF LABOUR	GCT+ GTT-VE (48)		GDM (15)	
	No	%	NO	%
SPONTANEOUS	30	62.5	5	33.3
INDUCED	18	37.5	10	66.7

Out of those delivered vaginally, 62.5% had spontaneous onset and 37.5% had induced labour in group I and 33.3% had spontaneous onset and 66.7% had induced labour in group II.

TABLE – 10

MODE OF DELIVERY

MODE OF DELIVERY	GCT+ GTT-(65)		GDM(28)	
	No	%	No	%
LABOUR NATURAL	30	46.2	10	31.7
VACUUM	18	27.7	4	14.3
FORCEPS	–	–	1	3.6
ELECTIVE C/S	7	10.8	3	10.7
EMERGENCY C/S	10	15.4	10	35.7



INDICATIONS FOR CAESEREAN SECTION

GROUP I :

ELECTIVE:

- Previous lscs with hydramnios – 1
- Previous lscs with CPD major – 3
- Previous lscs with BOH – 1
- Oligohydramnios with vulval warts – 1
- CPD major and macrosomia – 1

EMERGENCY:

- Non progression of labour – 6
- Fetal distress – 2
- Severe PIH with IUGR with fetal distress – 1
- Breech – 1

IN GROUP II:

ELECTIVE LSCS:

- Previous lscs with CPD – 1
- BOH – 1
- CPD major with macrosomia – 1

EMERGENCY LSCS:

- Nonprogression of labour – 5
- Fetal distress – 4
- Oligo hydramnios and fetal distress – 1

MATERNAL MORTALITY AND MORBIDITY – NIL

PERINATAL MORTALITY – NIL

PERINATAL MORBIDITY:

TABLE – 11

BIRTH WEIGHT

BIRTH WEIGHT	GCT+GTT-VE		GDM	
	No	%	No	%
<2	1	1.5	1	3.6
2 – 2.5	2	3.1	3	10.7
2.6 – 3	27	41.5	8	28.6
3.1 - 3.5	29	44.6	10	35.7
3.6 – 4	5	7.7	5	17.9
>4	1	1.5	1	3.6

- ⇨ Mean birth weight in group I – 3.09
- ⇨ Mean birth weight in GCT –VE – 2.94
- ⇨ Mean birth weight in group II – 3.01

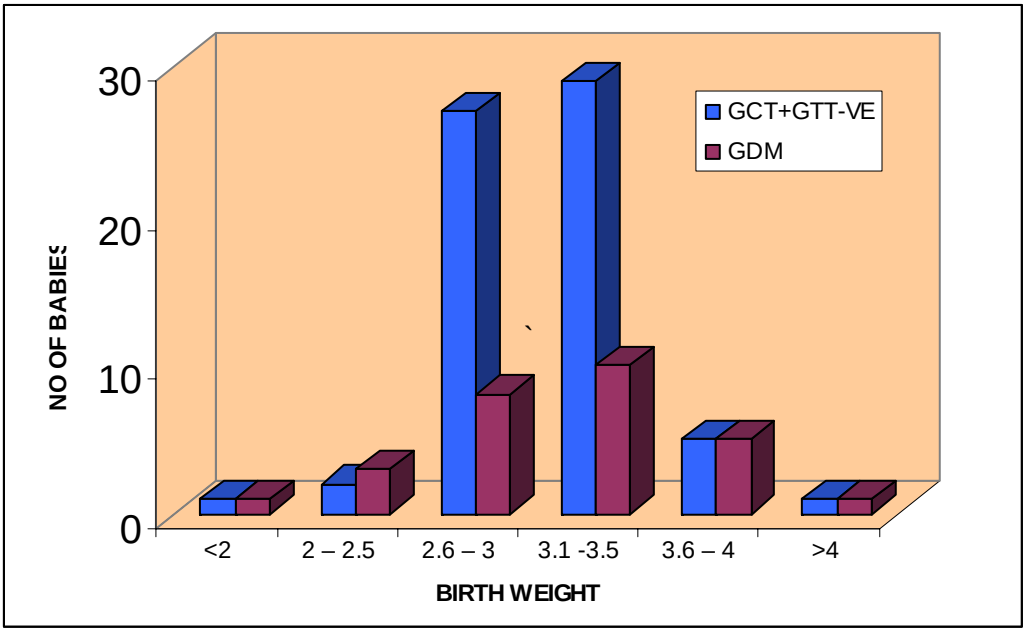


TABLE – 12

APGAR

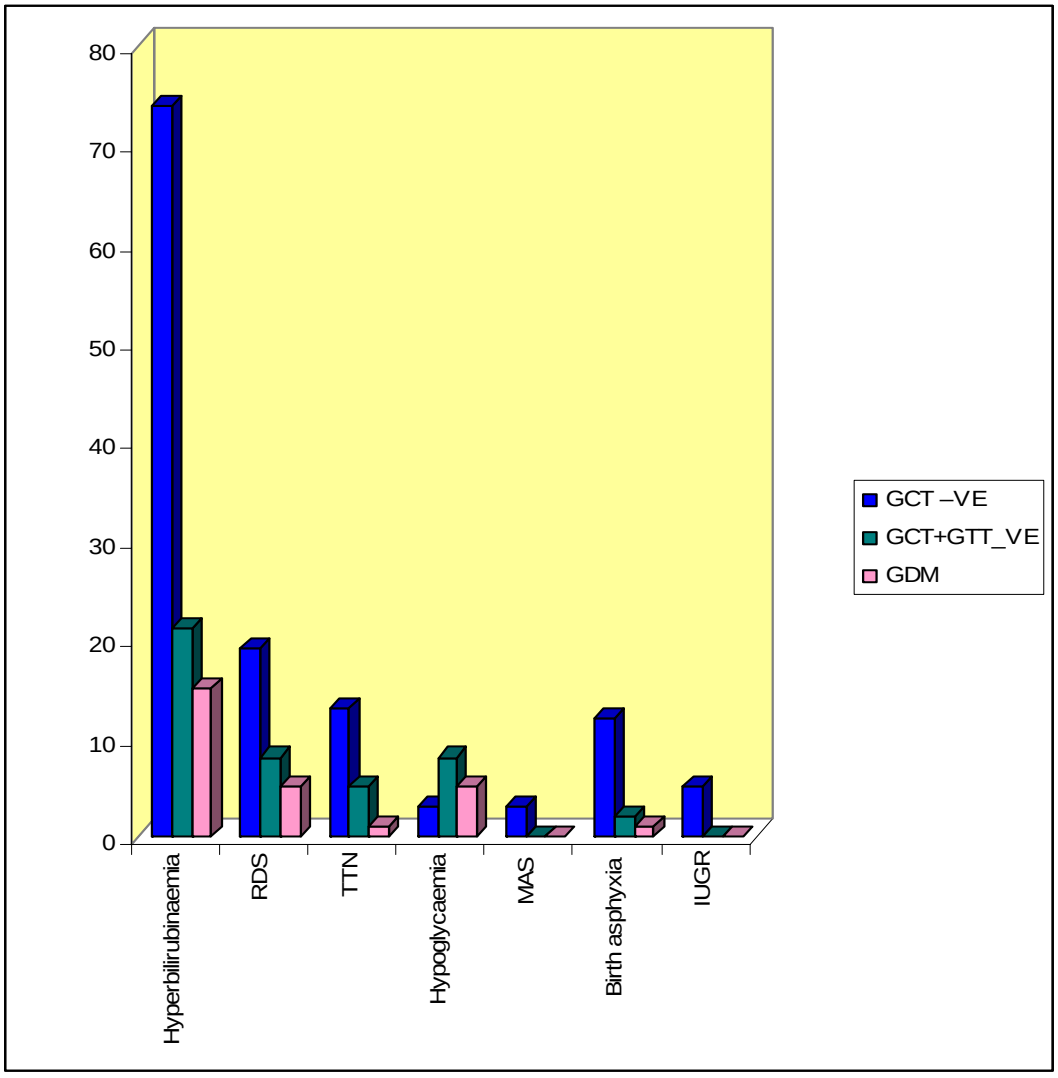
APGAR	GCT+GTT-VE		GDM	
	No	%	NO	%
<6/10	2	3	_	_
>7/10	63	97	28	100
5MT APGAR <6/10	_		_	_

PERINATAL OUTCOMES

NICU ADMISSION:

- ⇨ In group I, 31 (47.7%)babies got admitted to NICU.
- ⇨ In the group II,19(67.9%) babies got admitted in NICU.
- ⇨ In the control group,101 (24.8%) babies got admitted in NICU.

NICU ADMISSION	GCT–VE CASES(407)	GCT+GTT-VE (65)	GDM(28)
Hyperbilirubinaemia	**74**	**21**	**15**
RDS	**19**	**8**	**5**
TTN	**13**	**5**	**1**
Hypoglycaemia	**3**	**8**	**5**
Meconium Aspiration Syndrome	**3**	**-----**	**----**
Birth asphyxia	**12(1-severe)**	**2**	**1**
IUGR	**5**	**----**	



MACROSOMIA

- ⇨ In group I – 1.6% of babies had macrosomia
- ⇨ In group II – 3.6% of babies had macrosomia
- ⇨ In GCT – VE group, 0.5% of babies had macrosomia

SHOULDER DYSTOCIA

- ⇨ In group I – 1.6%
- ⇨ In group II – 3.6%
- ⇨ In GCT –VE – 0.25%

ERB'S PALSY

In group I, 2 babies had erb's palsy compared to none in group II and normal GCT group.

ANTENATAL DEATH

No antenatal deaths in both group I and group II groups compared to 3 in normal GCT group.

COMPARING THE DEMOGRAPHIC AND PERINATAL OUTCOMES

BETWEEN GCT+ GTT-VE AND GCT –VE GROUP

DEMOGRAPHIC PARAMETERS	GCT+GTT-VE	GCT -VE	t	P
MEAN AGE	27.2	25.6	3.15	0.01(SIGNIFICANT)
MEAN BMI	26.55	25.77	1.711	0.1(NOT SIGNIFICANT)
MEAN BIRTH WEIGHT	3.09	2.94	0.68	0.5(NOT SIGNIFICANT)

PERINATAL OUTCOME	GCT+GTT-VE (65)	GCT-VE (407)	ODDS RATIO	P
NICU ADMISSION	31 (47.7%)	101(24.8%)	2.76 (1.56 – 4.88)	<0.001(SIGNIFICANT)
MACROSOMIA	1(1.6%)	2(0.5%)	2.44 (0.49 – 12.3)	0.88 (NOT SIGNIFICANT)
SHOULDER DYSTOCIA	1(1.6%)	1(0.25%)	3.67 (0.9 – 14.9)	0.64 (NOT SIGNIFICANT)
ERB'S PALSY	2(3.2%)	0	7.46 (5.9 – 9.39)	0.018(SIGNIFICANT)
C/S	17(35.4%)	111(37.5%)	0.94 (0.5 – 1.77)	0.85(NOT SIGNIFICANT)
PREECLAMPSIA	8(12.3%)	26(6.38%)	2.06 (0.8 – 5.1)	0.07(NOT SIGNIFICANT)

From this study, it shows that higher the maternal age, more chances of developing glucose intolerance. Mean maternal age rates, NICU admission and erb's palsy rates are statistically significant (P<0.05) in GCT+GTT-VE group when compared to normal GCT-VE group.

GCT VALUES BETWEEN 130 – 140

No of patients with GCT values between 130–140 mg/dl–24. 4 babies born to these mothers were admitted in NICU, 2 had hyperbilirubinaemia, 1 had hypoglycaemia and 1 had

IUGR. Comparing this group with GCT <130mg/dl, odd's ratio is 0.59 (0.17–1.89). P=0.34 (>0.05% - not significant).

VALIDITY OF O'SULLIVANS GLUCOSE CHALLENGE TEST

500 unselected pregnant women were subjected to O'Sullivan glucose challenge test. 93 (18.6%) had values above 140 mgs%, among them 28 (30.1%) had GTT+ with O'Sullivan & Mahan GTT.

OGCT	+ OGCT	GDM
>140MGS/DL	**93 (18.6%)**	**28 (30.1%)**

CARLOTITI N et al conducted a mass screening for gdm using O'SULLIVAN GCT test in 751 women with 28 weeks of gestation. GCT positive in 18% of cases. Oral GTT confirmed the diagnosis in 14% of GCT positive cases. Compared with our study, where O'Sullivan GCT test was positive in 18.6%, oral GTT confirmed the diagnosis in 30.1% of these positive patients.

DISCUSSION

Gestational Diabetes Mellitus is one of the commonest medical complications of pregnancy. GDM is a risk factor for both mother and foetus. The risk increases proportionally to the to the maternal blood sugar concentration. So we have to screen all antenatal patients with glucose challenge test between 24 – 28 weeks of gestation and proceed with GTT if GCT is positive. Early diagnosis of GDM reduces perinatal morbidity and mortality.

In this study, incidence of GDM is 5.6% Incidence of GCT+GTT–VE patients was 13.77%. STAMILIO et al (2004) studied 1825 eligible pregnant women among a cohort of 1998 patients and identified 164 patients with GCT+GTT-VE and 50 patients with GDM, where the incidence of GCT+GTT-VE was 9.23%. MOSES et al (1995) performed GTT in 1185 women and found that GDM was present in 6.7% of women. GDM was present in 8.5 % of women aged more than 30 yrs.

In our study positive predictive value of GCT was 30.1%. JIMENEZ – MOLCON et al reported that, GCT has a positive predictive value of 12– 40%

In this study false +ve GCT was present in 5.9% of women aged <20yrs, 11.5% of women aged 21-30 yrs, 27.1% of women aged >31yrs.

GDM was present in 5.9% of women aged 21-30 yrs and 6.8% of women >31yrs. Most patients in both groups belonged to the age group 21–30 years and some patients were in the age group above 30 years showing that increase in age could lead to GDM and glucose intolerance.

In this study, mean age in GCT+GTT-VE group is 27.2 and in normal GCT–VE group is 25.6, which is statistically significant (P<0.01). In the study conducted by STAMILIO et al, mean age in GCT + GTT –ve group was 28.5 and in normal GCT –VE group was 25.5, which was statistically significant (P – 0.001)

In our study, GCT+ GTT-VE was present in 10.1% of primi, 12.4% of second gravida, 27.9% of third gravida and 5.3% of fourth gravida. GDM was present in 4.4% of primi, 6.7% of second gravida, 4.4% of third gravida and 15.8% of fourth gravida. In our study, in both groups, more % of women were found in third gravida. This shows that the more the number of pregnancies, the occurrence of glucose intolerance increases.

In our study, in GCT + GTT –VE group, 16.7% belong to II SES, 33.8 % to III SES, 8.5% to IV SES ,13.8 % to V SES. In GDM , 11.1% belong to SES II ,15.5% to SES III , 3.9% to SES IV ,3.4% to SES V. In both groups, more % of women belong to higher socioeconomic strata.

In this study, GCT+GTT–VE results were present in 16.7 % of women with BMI < 20, 10.5% of women with BMI of 21–25, 13.5% of women with BMI of 26 – 30, 17.4 % of women

with BMI >30. GDM was present in 2.8% of women with BMI 20 – 25, 4.9% of women with BMI 26 – 30,15.9% of women with BMI >30.

In our study, more no of women were found with BMI above 26 showing that glucose intolerance and BMI are directly proportional to each other. In our study, mean BMI in GCT+GTT-VE is 26.55 and in normal GCT –VE is 25.77, which is not statistically significant (P – 0.1).

In the study conducted by STAMILIO et al, mean BMI WAS 28.5 in GCT+GTT-VE group and 25.5 in normal GCT–VE group, which was significant (P<0.001).

In this study, in GCT+VE GTT–ve group, 32.1% had family h/o DM in father, 22.2% had family h/o DM in the mother and 33.3% in both parents. In GDM, 14.3% had DM in the father, 22.2% in the mother, 33.3% in both the parents.

MOSES et al showed that GDM was present in 11.6% with positive family history. According to GARNER el al (1995) if mother had IDDM, 1–3% of offsprings developed diabetes, if father had IDDM, 6% developed diabetes and with positive H/O DM in both parents 20% developed diabetes. Hence a H/o IDDM in the father is of greater predictive value than in the mother and even greater when a sibling is diabetic. A family history in grand parents is less significant. Family H/O diabetes either in the first degree or in the second degree relative is an important risk factor for developing abnormal glucose tolerance (5 – 10 times risk greater than for children with non diabetic parents).

In this study, urine sugar was positive in 7 patients out of 500. In the GCT+GTT-group, 28.6% had positive urine sugar. In GDM, 42.9% had positive urine sugar.

Urine sugar is not a sensitive test for identifying patients with glucose intolerance. Most pregnant patients have renal glycosuria as a result of low threshold for elimination of glucose by the kidneys and have normal kidneys.

In this study, % of PIH in GCT+ GTT- is 12.3% and % of GDM and PIH -7.1%. According to STAMILIO et al (2004), 6.5% of patients with GCT+ GTT- had PIH. According to GABBE et al (1997) and JACOBSON & COUSIN (1989),the rate of PIH among GDM subjects is not significantly different from controls. GARNER et al & ROSSEN et al have shown that PIH was twice high in GDM.

In this study, % of hydramnios in women with GCT+GTT-VE – 3.1% and % of hydramnios in patients with GDM -7.1%. JACOBSON & COUSIN et al reported an incidence of hydramnios in GDM patients as 2% compared with our study where incidence was 3.1% in GCT+GTT- patients and 7.1% in GDM. Hydramnios affects approximately 0.4% 1.5 % of all pregnancies. Diabetes mellitus may be responsible for approximately 14% of all cases of polyhydramnios. In our study, the incidence of hydramnios is only 7.1% in GDM, showing that the diabetic status during pregnancy was well controlled.

In GCT + GTT-VE cases, preterm labour occurred in 2(3.1%) and in GDM, preterm labour occurred in 1(3.6%). Incidence of preterm labour in GDM as per 1986 – 1993 review was 7.3% and 20 – 40% in overt diabetes compared with our study where incidence of preterm labour in GCT+GTT- cases were 3.1% and in GDM, it is 3.6%. the incidence of preterm labour is more in overt DM than in GDM and GCT+GTT- because of increased risks of infections like chorioamnionitis precipitating preterm labour.

In GCT + GTT –VE, there were previous history of 2 neonatal deaths (1 due to severe PIH), 2 anencephaly, 1 hydrocephalus and 1 anomalous baby (diaphraghmatic hernia). In all these patients, probably GDM was missed in previous pregnancy. In GDM, there were

previous history of 2 neonatal deaths, 2 IUD (1 was due to severe PIH), 1 patient had H/O GDM in previous pregnancy and macrosomia – 1 (4.2 kgs)

In our study,13.3% and 7.1% delivered at term and preterm respectively in GCT+GTT-VE group and 5.7% and 3.6% delivered at term and preterm respectively.

20 out of 28 (71.4%) were treated with mealplan till delivery. 8 out of 28 (28.6%) were switched from mealplan to insulin due to poor glycemic control, till delivery. According to ACOG technical bulletin, 10 – 15% of patients with GDM require insulin.

In our study, in GCT+GTT-VE group, 46.2% delivered normally, 27.7% delivered by vacuum, 10.8% by elective LSCS and 15.4% by emergency LSCS being done for macrosomia in 1.5%. In GDM group, 35.7% delivered normally, 14.3% delivered by vacuum, 3.6% by forceps, 10.7% by elective LSCS, 35.7% delivered by emergency LSCS being done for macrosomia in 3.6%. COUSTAN and EMARAH et al (1994) in a retrospective study of 299 cases found that caeserean section was done in 44.4% of cases, being done for macrosomia in 7% of cases.

Eventhough LSCS rate is high in GCT+GTT-VE group, it is not statistically significant (P=0.85) when compared to normal GCT group, where the LSCS rate was 37.5% because of increased previous LSCS rates.

In the immediate postpartum period, once the patients resumed normal diet, fasting and postprandial blood sugar was checked in GDM. Out of 28, 3 had elevated FBS and PPBS (10.7%).

Mean birth weight in GCT+GTT –VE patients – 3.09, mean birth weight in GCT –VE patients – 2.94 and mean birth weight in GDM – 3.01. There were about 41.5% of babies

weighing between 2.6 – 3 kgs, 44.6% of babies weighing between 3.1–3.5 kgs, 7.7% of babies between 3.6–4 kgs, 1.5% >4 kgs, 1.5% was <2kgs and 3.1% were between 2 – 2.5 kgs in GCT+GTT-VE group.

In the GDM group, 28.6% of babies weighed between 2.6 – 3 kgs, 35.7% of babies weighed between 3.1 - 3.5 kgs, 17.9% were between 3.6 – 4 kgs and 3.6% were > 4 , 3.6% were less than 2 kgs and 10.7% were between 2 -2.5 kgs.

In the STAMILIO et al (2004) study, mean birthweight is 3.304 kgs in GCT+GTT-patients and 3.194 kgs in normal GCT patients. Because of the treatment given to GDM, incidence of macrosomia is very less in our study group. In our study, when comparing mean birth weight between GCT+GTT-VE and normal GTT group, it is not statistically significant (P=0.5).

In our study, in GCT +GTT-VE group, 1 mt apgar was >7/10 in 63 (97%) of babies and 2(3%) babies had apgar <6/10. In these babies 5 mt apgar was above 7/10. In GDM group, all babies had apgar >7/10.

All babies in GDM group had good apgar because of good glucose control and prompt resuscitation.

Maternal mortality was nil.

Perinatal and neonatal mortality was nil

In GCT +GTT-VE group, 47.7% babies got admitted to NICU. In the GDM group, 67.85% babies got admitted in NICU. In the control group, 24.8% babies got admitted in NICU. Factors taken into account for NICU admission were hyperbilirubinaemia, hypoglycaemia, respiratory distress syndrome, meconium aspiration syndrome, transient tachypnoea of

newborn, birth asphyxia and IUGR. When comparing GCT+GTT-VE group and control group, NICU admission is statistically significant in GCT+GTT-VE (P <0.001).

In the study by STAMILIO et al,14 babies (8.6%) got admitted to NICU. In the normal GCT group, 163 babies (9.8%) got admitted (statistically not significant, P – 0.61).

REY et al reported that patients with an abnormal GCT and a single elevated value on the GTT are at increased risk for fetal macrosomia, neonatal hypoglycaemia and neonatal hyperbilirubinaemia.

KHAN et al reported that patients with a positive glucose screening test and a negative GTT were at increased risk for fetal macrosomia, caeserean delivery and pre-eclampsia.

In GCT+GTT–VE group, 1.6% baby had macrosomia,in GDM, 3.6% baby had macrosomia, in GCT–VE group, 0.5% babies had macrosomia, (statistically not significant, P-0.88). In the STAMILIO et al study, 14 babies (8.5%) had macrosomia in the GCT+GTT- and 95 babies (5.7%) in the normal GCT group, (statistically not significant, P – 0.15).

Using a case control study design, OKUN et al showed that patients with an abnormal GCT and no elevated values on the GTT are at increased risk for foetal macrosomia. The incidence of macrosomia is less in GDM because of good glycaemic control in pregnancy.

In GCT+GTT-VE group, 1.6% baby had shoulder dystocia, in GDM, 3.6% baby had shoulder dystocia and in GCT –VE, 0.25% baby had shoulder dystocia. When comparing these two groups it is not statistically significant (P= 0.64).

In the study by STAMILIO et al, shoulder dystocia was present in 8 babies (4.9%) in GCT +GTT-VE group and in 29 babies (1.7%) in normal GCT group, statistically significant (P=0.007).

In GCT+GTT-VE group,2 babies had erb's palsy compared to none in GDM and normal GCT group (statistically significant, P.018).

35.4% babies delivered by C/S in GCT +GTT-VE group compared to 37.5% in normal GCT group. 46.4% babies delivered by C/S in GDM. In the study by STAMILIO et al, 18 babies (23.8%) were delivered by caeserean section in GCT +GTT-VE group and 286 babies (17.2%) were delivered by caeserean section in normal GCT group.

No antenatal deaths in both GCT=GTT-VE and GDM groups compared to 3 in normal GCT group. In the study by STAMILIO et al, there were 1.2% antenatal death in GCT+GTT-VE group and 6 (0.4%) in normal GCT group.

From this study, it shows that higher the maternal age, more chances of developing glucose intolerance. Mean maternal age rates, NICU admission and erb's palsy rates are statistically significant($P<0.05$) in GCT+GTT-VE group when compared to normal GCT-VE group.

In the study by STAMILIO et al, mean BMI, mean maternal weight, mean birth weight rates were statistically significant ($P<0.05$) in GCT+ GTT-VE group when compared to normal GCT group. Shoulder dystocia and caeserean delivery are significant in GCT+GTT-VE group when compared to normal GCT group.

No of patients with GCT values between 130 – 140 mg/dl were 24. 4 babies born to these mothers were admitted in NICU,2 had hyperbilirubinaemia, 1 had hypoglycaemia and 1 had IUGR. Comparing this group with GCT <130mg/dl, odd's ratio is 0.59 (0.17–1.89). P=0.34

(>0.05% -not significant). So according to this study, we need not subject patients with GCT <140 mgs/dl to GTT.

500 unselected pregnant women were subjected to O'Sullivan glucose challenge test. 93 (18.6%) had values above 140 mgs%, among them 28 (30.1%) had GTT+ with O' Sullivan & Mahan GTT.

CARLOTITI N et al conducted a mass screening for GDM using O'Sullivan GCT test in 751 women with 28 weeks of gestation. GCT positive in 18% of cases. Oral GTT confirmed the diagnosis in 14% of GCT positive cases. Compared with our study, where O'Sullivan GCT test was positive in 18.6%, oral GTT confirmed the diagnosis in 30.1% of these positive patients.

CONCLUSION

Of 500 patients taken up for the study, incidence of GDM in our study is 5.6%. Incidence of GCT+GTT–VE patients was 13.77%. OGTT is the gold standard in the diagnosis of abnormal glucose tolerance during pregnancy and perhaps is desirable even as a screening test. Owing to practical difficulties, O'Sullivan glucose challenge test is a convenient, economical and suitable alternative screening test without sacrificing sensitivity expected of a screening test. Positive predictive value of GCT in our study was 30.1%.

False positive GCT (GCT+GTT-VE) is identified as an independent risk factor for adverse perinatal outcome, including the maternal outcome variable, NICU admission, shoulder dystocia, foetal macrosomia, caesarean delivery and antenatal death. So identifying these patients is important, so that we can monitor them well and can prevent complications. They could benefit from additional therapies, such as more intensive foetal monitoring, nutritional counselling or a diabetic diet.

From this study, it shows that higher the maternal age, more chances of developing glucose intolerance. Mean maternal age rates, NICU admission and erb's palsy rates are significant ($P<0.05$) in GCT+GTT-VE group when compared to normal GCT-VE group.

If the GCT <140 mg/dl, they can be treated as normal.

In the case of macrosomia occuring in the normal GCT group, patients are advised to do postnatal fasting and postprandial blood sugar.

As per the recommendations of the international workshop conference of gestational diabetes, all pregnant women should be screened for gestational diabetes at 24–28 weeks of gestation by O' Sullivan's 50 gms glucose challenge test. It is important for two reasons. First,

identification of women with GDM followed by appropriate treatment and monitoring will reduce foetal macrosomia and identify those women at risk for foetal macrosomia. Second, given the high likelihood that women who manifest GDM will develop type II. Identification of these patients will permit intervention after delivery that might delay or prevent the onset of type -II Diabetes Mellitus.

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PROFORMA

ROUTINE ANTENATAL SCREENING WITH GLUCOSE CHALLENGE TEST AND ITS RELATION WITH PERINATAL OUTCOME

Name :

Age :

SES :

Education :

Gravida : **Para :** **Livebirth:**

Abortions:

LMP :

EDD :

Menstrual History:

Obstetric History:

Past History:

Family History:

Examination:

Date	Height	Weight	B.P	Pedal edema	P/A	Expected uterus size

Investigations:

Urine sugar :

GCT :

GTT :

USG :

HbA1C :

(in mother with poor perinatal outcome)

In GTT +ve, periodical blood sugar F & PP :

Treatment: Diet / insulin / OHA

If on Insulin drug dose :

GA at time of delivery :

Mode of delivery : (1) Vaginal (2) Elective LSCS (3) Emergency LSCS

If vaginal: Spontaneous / induced

If induced: Mode of induction

I stage of labour:

II stage of labour:

III stage of labour:

<u>Baby :</u> Liveborn / Deadborn

If live born APGAR:

Weight:

NICU admission:

If deadborn **Fresh death**

Macerated

Maternal complications:

Postnatal blood sugar in GDM:

MASTER CHART

No.	Name	Age	SES	Obst. Score	GA	Obs. History	Risks	Fam History	BMI	PN Bld. Sugar	Urine Sugar	GCT	GTT	Periodi Cal Bld. Sugar	Diet Insul
01.	Shanthi	29	II	$G_3P_1L_4A_1$	T	Pr CS	-	-	26.04	-	-	163	N	-	-
02.	Vanitha	27	IV	Primi	T	-	-	-	27.43	-	-	142	N	-	-
03.	Shabita	27	III	$G_3P_2L_4$	T	Anen.	-	-	30.42	-	-	147	N	-	-
04.	Nirmala	20	IV	G_2A_1	T	-	-	F	27.12	-	-	140	N	-	-
05.	Mahes	22	III	G_2A_1	T	-	GDM	F	26.08	N	-	153	GDM	N	D
06.	Padma	21	IV	Primi	T	-		-	26.06	-	-	108	-	-	-
07.	Sumathi	22	IV	P	T	-	PIH	-	30.89	-	-	80	-	-	-
08.	Jeeja	30	III	$G_2P_1L_4$	T	-		-	26.29	-	-	112	-	-	-
09.	Amuda	32	III	$G_3P_1L_4A_1$	T	Pr CS	Oligo	-	33.33	-	-	100	-	-	-
10.	Lakhmi	26	IV	Primi	PT	-	PIH	-	29.51	-	-	125	-	-	-
11.	Chitra	27	IV	$G_2P_1L_0$	T	Pr SB	-	-	27.92	-	-	107	-	-	-
12.	Jaya	27	IV	$G_3P_1L_4A_1$	T	-	-	-	25.75	-	-	108	-	-	-
13.	Shanthi	20	III	Primi	T	-	-	F	24.55	-	-	134	-	-	-
14.	Sathya	28	III	Primi	T	-	-	-	31.84	-	-	116	-	-	-
15.	Vincy	24	IV	$G_3P_2L_2$	T	-	-	-	25.28	-	-	106	-	-	-
16.	Vijaya	22	IV	Primi	PT	-	-	F	20.17	-	-	118	-	-	-
17.	Deivani	23	V	$G_2P_1L_0$	T	Pr CS	-	-	23.11	-	-	110	-	-	-
18.	Mahes	29	IV	$G_3P_1L_1A_1$	T	Pr CS	-	-	27.02	-	-	108	-	-	-
19.	Akila	28	III	Primi	T	-	-	-	26.07	-	-	88	-	-	-
20.	Indira	28	IV	$G_4P_2L_1A_1$	T	Pr CS	-	-	26.20	-	-	136	-	-	-
21.	Devika	23	IV	$G_4P_2L_1A_1$	T	-	-	-	41.53	-	-	134	-	-	-
22.	Chitra	30	III	$G_2P_1L_1$	T	-	-	-	24.06	-	-	116	-	-	-
23.	Kala	24	IV	$G_4P_1L_4A_2$	T	-	-	-	26.05	-	-	120	-	-	-
24.	Gandhi	23	IV	Primi	T	-	-	-	21.41	-	-	77	-	-	-
25.	Bagya	23	IV	Primi	T	-	-	-	26.11	-	-	112	-	-	-
26.	Saranya	29	IV	$G_2P_1L_1$	T	Pr CS	-	-	27.12	-	-	116	-	-	-
27.	Sasi	20	IV	Primi	T	Oligo.	-	-	25.67	-	-	77	-	-	-
28.	Prema	21	V	P	T	-	PIH	-	22.08	-	-	80	-	-	-
29.	Bagyla	26	III	$G_2P_1L_1$	T	Pr CS	-	-	23.62	-	-	100	-	-	-
30.	Sudha	26	III	$G_4P_2L_1A_1$	T	Pr CS	-	-	26.29	-	-	101	-	-	-
31.	Rajmani	27	IV	Primi	T	Pr CS	-	-	21.05	-	-	103	-	-	-
32.	Revathy	21	V	Primi	T	-	-	-	20.95	-	-	63	-	-	-
33.	Carolin	27	IV	$G_2P_1L_1$	T	-	PROM	-	25.56	-	-	81	-	-	-
34.	Shantha	38	IV	$G_2P_1L_1$	T	-	PIH	M	25.63	-	-	141	N	-	-
35.	Anurda	26	III	$G_2P_1L_1$	T	-	-	-	24.38	-	-	132	-	-	-
36.	Rabiya	23	IV	Primi	T	-	-	-	27.12	-	-	116	-	-	-
37.	Lavana	26	III	Primi	T	-	-	-	22.65	-	-	118	-	-	-
38.	Sabitha	23	III	$G_3P_1L_1A_1$	T	-	-	-	24.03	-	-	134	-	-	-
39.	Sunitha	23	III	Primi	T	-	Oligo	-	24.52	-	-	83	-	-	-
40.	Priya	22	III	Primi	T	PROM	-	-	18.55	-	-	90	-	-	-
41.	Kiritika	26	IV	Primi	T	-	-	-	26.28	-	-	130	-	-	-
42.	Sulo	27	III	$G_2P_1L_1$	T	-	-	F	29.23	-	-	146	N	-	-
43.	Rani	23	III	Primi	T	-	-	-	27.12	-	-	124	-	-	-
44.	Subha	25	IV	G_3A_2	T	-	-	-	31.24	-	-	69	-	-	-
45.	Vasatha	28	IV	$G_2P_1L_1A_1$	T	Pr CS	-	M	28.57	-	-	90	-	-	-
46.	Kavitha	30	III	$G_2P_1L_1$	T	-	-	-	24.88	-	-	112	-	-	-
47.	Gowsal	26	IV	Primi	T	-	-	-	21.11	-	--	69	-	-	-
48.	Kavitha	25	IV	Primi	PT	-	HIV+	-	22.31	-	-	90	-	-	-
49.	Rani	30	IV	$G_2P_1L_1A_1$	T	-	-	-	24.45	-	-	101	-	-	-
50.	Kumua	25	III	$G_2P_1L_1$	T	Pr CS	-	-	20.77	-	-	112	-	-	-
51.	Naga	30	IV	Primi	T	-	-	-	19.90	-	-	108	-	-	-
52.	Patmal	27	III	Primi	T	-	-	-	25.14	-	-	118	-	-	-

53.	Neela	30	IV	$G_2P_1L_1$	T	-	-	-	24.09	-	-	120	-	-	-
54.	Mena	20	III	Primi	T	-	-	-	24.34	-	-	79	-	-	-
55.	Parvaty	25	IV	Primi	T	-	PROM	-	27.43	-	-	89	-	-	-
56.	Karuna	34	IV	$G_2P_1L_1A_1$	T	-	-	-	24.53	-	-	112	-	-	-
57.	Kala	24	IV	$G_2P_1L_1$	T	-	-	-	23.62	-	-	108	-	-	-
58.	Malar	25	V	$G_6P_2L_2A_3$	PT	-	-	-	17.62	-	-	95	-	-	-
59.	Sahar	28	V	Primi	PT	-	Oligo	-	18.90	-	-	100	-	-	-
60.	Shantha	23	IV	$G_2P_1L_1$	T	-	-	-	21.87	-	-	119	-	-	-
61.	Shanthi	28	IV	$G_2P_1L_1$	T	-	VVB	-	23.92	-	-	72	-	-	-
62.	Chitra	26	III	P	T	-	-	MF	25.76	-	-	140	-	-	-
63.	Malathy	23	III	P	T	-	-	-	25.28	-	-	142	-	-	-
64.	Mallika	32	IV	P	T	-	-	M	26.47	-	+	162	N	-	-
65.	Anita	19	III	P	T	-	-	-	25.63	-	-	181	-	-	-
66.	Chitra	23	III	P	T	-	-	-	23.93	-	-	140	-	-	-
67.	Parvaty	23	III	P	T	-	Epilep	-	27.76	-	-	153	-	-	-
68.	Dowlath	28	V	P	T	-	GDM	-	30.26	-	-	145	N	D	N
69.	Rajesh	22	IV	P	T	-	GDM	-	22.22	-	-	150	N	D	N
70.	Sugnya	22	III	P	T	-	GDM	M	32.79	-	-	146	N	D	N
71.	Baby	28	IV	P	T	-	Rh	-	27.30	-	-	123	-	-	-
72.	Sujatha	30	IV	$G_2P_1L_1$	T	-	GDM	-	35.6	-	-	114	-	-	-
73.	Jeyama	27	IV	Primi	T	-	Oligo	-	25.96	-	-	122	-	-	-
74.	Malathy	27	III	P	PT	-	Oligo	-	25.47	-	-	100	-	-	-
75.	Vanitha	25	III	$G_2P_1L_1$	T	-	TMS	-	31.93	-	-	92	-	-	-
76.	Sunitha	28	III	P	T	-	TMS	F	27.88	-	-	117	-	-	-
77.	Vijaya	22	IV	$G_2P_1L_1$	T	Pr CS	Polio	-	20.92	-	-	83	-	-	-
78.	Adhi	34	IV	$G_2P_1L_1$	T	Pr CS	Oligo	-	24.12	-	-	86	-	-	-
79.	Amsa	25	IV	Primi	T	-	-	F	26.10	-	-	98	-	-	-
80.	Selvi	25	IV	$G_2P_1L_1$	T	Pr CS	-	-	26.63	-	-	101	-	-	-
81.	Karpam	31	III	$G_3P_1L_1A_1$	T	Pr CS	-	-	31.93	-	-	140	N	-	-
82.	Vimala	28	V	$G_2P_1L_1$	T	Pr CS	-	-	19.94	-	118	-	-	-	-
83.	Saras	33	III	$G_3P_1L_1A_1$	T	Rh -	-	-	26.02	-	136	-	-	-	-
84.	Mohana	25	II	Primi	T	Oligo	-	-	19.47	-	111	-	-	-	-
85.	Maha	33	V	$G_2P_1L_1A_1$	T	Pr CS	-	-	34.01	-	114	-	-	-	-
86.	Rani	23	IV	$G_3P_1L_1A_1$	T	Pr CS	-	-	27.63	-	103	-	-	-	-
87.	Kavitha	34	III	Primi	T	-	-	-	37.09	-	118	-	-	-	-
88.	Sudha	20	IV	Primi	T	Oligo	-	-	27.77	-	111	-	-	-	-
89.	Lakshm	29	IV	$G_2P_1L_1$	T	-	-	-	26.84	-	75	-	-	-	-
90.	Raja	18	IV	Primi	T	Oilgo	-	-	20.08	-	105	-	-	-	-
91.	Velu	30	III	$G_2P_1L_1$	T	-	-	-	25.86	-	111	-	-	-	-
92.	Radha	22	IV	Primi	T	PROM	-	-	24.83	-	96	-	-	-	-
93.	Vijaya	28	V	$G_2P_1L_1$	T	-	-	-	26.67	-	150	N	-	-	-
94.	Sumathi	28	IV	$G_2P_1L_1$	T	Oligo	-	-	31.55	-	109	-	-	-	-
95.	Mythili	20	IV	G_2A_1	T	Enc	-	-	19.36	-	109	-	-	-	-
96.	Siranje	27	IV	Primi	T	-	-	-	23.78	-	85	-	-	-	-
97.	Nalini	22	IV	$G_2P_1L_1$	T	-	-	-	26.29	-	132	-	-	-	-
98.	Dhana	21	IV	Primi	T	-	-	-	22.18	-	162	N	-	-	-
99.	Ratinam	24	IV	G_2A_1	T	-	-	-	27.84	-	122	-	-	-	-
100	Stella	32	IV	$G_2P_1L_1$	T	Pr CS	-	-	26.17	-	107	-	-	-	-
101.	Kancha	27	IV	Primi	T	-	-	-	23.1	-	110	-	-	-	-
102.	Sneha	29	IV	$G_2P_1L_1$	T	-	PIH	F	24.8	-	106	-	-	-	-
103.	Padma	21	IV	G_2A_1	T	-	-	-	26.7	-	98	-	-	-	-
104.	Lakshi	29	IV	$G_2P_1L_1$	T	-	-	-	28.1	-	98	-	-	-	-
105.	Sudha	22	IV	$G_2P_1L_1$	T	-	-	-	21.0	-	94	-	-	-	-
106.	Kavitha	28	IV	$G_2P_1L_1A_1$	T	-	PROM	-	36.9	-	100	-	-	-	-
107.	Bhuva	26	V	$G_2P_1L_1$	T	-	-	-	26.3	-	98	-	-	-	-
108.	Mercy	23	III	Primi	T	-	-	-	24.0	-	104	-	-	-	-
109.	Pinky	29	IV	G_2A_1	T	-	Oligo	-	34.5	-	106	-	-	-	-
110.	Mahila	29	IV	$G_2P_1L_1A_1$	T	Pr CS	-	-	27.0	-	110	-	-	-	-
111.	Shantha	29	IV	$G_2P_1L_1$	T	-	-	-	24.8	+	164	N	-	-	-
112.	Nalini	25	III	$G_2P_1L_1$	T	-	-	-	23.1	-	140	N	-	-	-
113.	Sumaty	22	IV	Primi	T	-	Oligo	-	24.0	-	148	N	-	-	-
114.	Amuda	24	IV	Primi	T	-	-	-	23.96	-	148	N	-	-	-
115.	Sagaya	32	IIII	$G_2P_1L_1$	T	-	PROM	-	25.97	-	140	N	-	-	-
116.	Anita	24	III	Primi	T	-	Oligo	-	23.3	-	146	N	-	-	-
117.	Parmsh	28	III	$G_4P_2L_2A_1$	T	-	GDM	-	26.5	-	150	+	N	N	D

118.	Kavitha	32	III	$G_2P_1L_1$	T	Pr CS	GDM	-	29.6	-	148	+	N	N	D
119.	Sangeta	22	III	$G_2P_1L_0$	T	ND	GDM	-	26.92	++	249	+	N	↑	D
120.	Rukmai	26	IV	$G_2P_1L_1$	PT	-	-	-	28.2	-	91	-	-	-	-
121.	Anita	19	III	G_2A_1	T	-	PROM	-	23.6	-	100	-	-	-	-
122.	Preeti	27	IV	G_2A_1	T	-	-	F	28.0	-	114	-	-	-	-
123.	Selvam	29	III	Primi	T	-	-	-	26.5	-	112	-	-	-	-
124.	Priya	19	III	Primi	T	-	-	-	21.5	-	98	-	-	-	-
125.	Bhuva	27	III	Primi	T	-	-	-	28.1	-	114	-	-	-	-
126.	Shakita	25	V	G_2A_1	T	-	-	-	25.96	-	94	-	-	-	-
127.	Valar	27	IV	$G_2P_1L_1$	T	Pr CS	-	-	24.88	-	102	-	-	-	-
128.	Judy	21	III	Primi	T	-	-	-	26.34	-	133	-	-	-	-
129.	Celin	30	IV	$G_3P_2L_0$	T	-	-	-	32.88	-	105	-	-	-	-
130.	Radhai	26	III	$G_3P_1L_1A_1$	T	Pr CS	-	-	27.20	-	104	-	-	-	-
131.	Sangeta	18	III	$G_2P_1L_1$	T	Pr CS	-	-	30.78	-	100	-	-	-	-
132.	Synthia	29	V	Primi	T	-	-	-	21.11	-	126	-	-	-	-
133.	Kala	21	V	$G_2P_1L_0$	T	-	-	-	27.82	-	126	-	-	-	-
134.	Anbu	30	IV	$G_2P_1L_1$	T	Pr CS	-	-	31.24	-	99	-	-	-	-
135.	Srekala	27	IV	G_2A_1	T	-	-	-	24.03	-	128	-	-	-	-
136.	Kowsla	27	IV	Primi	T	-	-	-	27.47	-	98	-	-	-	-
137.	Shanthi	31	IV	$G_2P_1L_1$	T	-	-	-	26.29	-	100	-	-	-	-
138	Shanthi	26	IV	$G_2P_1L_1$	T	-	-	-	26.98	-	106	-	-	-	-
139.	Revathy	32	IV	G_2A_1	T	HC	PIH	-	35.15	-	146	N	-	-	-
140.	Vasanta	29	V	Primi	PT	PIH	Oligo	F	25.75	-	208	N	-	-	-
141.	Gomati	19	IV	Primi	T	-	-	-	34.23	-	106	-	-	-	-
142.	Jothi	24	IV	$G_2P_1L_1$	T	-	-	-	24.14	-	95	-	-	-	-
143.	Anita	20	IV	G_3A_2	T	-	-	-	21.77	-	100	-	-	-	-
144.	Nisreen	28	IV	$G_2P_1L_1$	T	-	PROM	F	30.01	-	158	N	-	-	-
145.	Lashmi	27	IV	$G_3P_1L_1A_1$	T	-	-	-	24.43	-	108	-	-	-	-
146.	Jaya	27	III	Primi	T	-	-	-	32.32	-	125	-	-	-	-
147.	Kala	25	IV	Primi	T	-	-	-	26.63	-	108	-	-	-	-
148.	Radhika	19	V	Primi	T	-	-	-	24.83	-	110	-	-	-	-
149.	Revathi	34	III	G_2A_1	T	-	PROM	-	35.15	-	123	-	-	-	-
150.	Vanitha	22	III	G_2A_1	T	-	-	-	24.88	-	112	-	-	-	-
151.	Kavitha	23	III	Primi	T	-	-	-	20.82	-	141	N	-	-	-
152.	Jayanti	23	III	P	T	-	-	-	18.05	-	101	-	-	-	-
153.	Sathya	21	III	P	T	-	-	-	29.17	-	128	-	-	-	-
154.	Lumee	23	III	P	T	-	PIH	-	31.97	-	91	-	-	-	-
155.	Bhaga	20	IV	P	T	-	-	-	21.11	-	106	-	-	-	-
156.	Sumaty	25	V	$G_3P_1L_1A_1$	T	Pr CS	-	-	22.21	-	154	N	-	-	-
157.	Nirmala	33	IV	Primi	T	-	-	-	23.55	-	106	-	-	-	-
158.	Megala	20	IV	Primi	T	IUGR	-	-	24.11	-	116	-	-	-	-
159.	Malathi	29	III	$G_3P_1L_1$	T	Oligo	-	-	19.62	-	106	-	-	-	-
160.	Anita	23	IV	Primi	T	-	-	-	23.83	-	110	-	-	-	-
161.	Vanitha	25	III	Primi	T	-	Oligo	-	25.87	-	108	-	-	-	-
162.	Kalpana	23	V	Primi	T	-	-	-	20.56	-	74	-	-	-	-
163.	Jothi	24	IV	$G_3P_1L_1$	T	-	-	-	24.99	-	95	-	-	-	-
164.	Jayanti	22	III	Primi	T	-	-	-	18.97	-	101	-	-	-	-
165.	Jenita	27	IV	$G_3P_1L_1$	T	-	-	-	28.56	-	108	-	-	-	-
166.	Jayanti	29	III	$G_4P_1L_1A_2$	T	Pr CS	-	-	25	-	120	-	-	-	-
167.	Anita	21	V	Primi	T	-	-	-	25.9	-	117	-	-	-	-
168.	Sakila	24	V	$G_3P_1L_1$	T	-	-	-	23.4	-	108	-	-	-	-
169.	Gayatiri	28	V	$G_2P_1L_1$	T	-	-	-	26.77	-	132	-	-	-	-
170.	Bagya	32	IV	Primi	T	-	-	PROM	27.74	-	146	N	-	-	-
171.	Amsa	23	III	$G_3P_1L_1A_1$	T	-	-	-	25.10	-	124	-	-	-	-
172.	Sarala	20	IV	Primi	T	Pr CS	-	-	37	-	103	-	-	-	-
173.	Subbu	21	III	$G_3P_1L_1$	PT	PIH	PIH	-	25.56	-	112	-	-	-	-
174.	Karupa	26	V	$G_3P_1L_1$	T	Pr CS	-	-	27.12	-	112	-	-	-	-
175.	Girija	24	IV	Primi	T	-	-	-	25.78	-	88	-	-	-	-
176.	Vijaya	31	III	$G_3P_1L_1A_1$	T	-	Epile	-	27.12	-	152	N	N	-	-
177.	Rajesh	30	V	$G_2P_1L_1$	T	-	-	-	26.56	-	131	N	N	-	-
178	Surya	31	IV	$G_4P_1L_1A_2$	T	-	-	F	28.44	-	152	+	N	D	-
179.	Christi	29	IV	$G_2P_1L_1$	T	Pr CS	PIH	-	36.96	-	158	+	N	I	N
180	Shanthi	26	IV	$G_2P_1L_0$	T	IUDPrS	-	-	32.54	-	192	+	N	D	N
181.	Kavitha	23	III	Primi	T	-	-	F	26.67	-	146	+	N	D	N

182.	Uma	30	IV	$G_2P_1L_1$	T	Pr CS	-	-	25	-	110	-	-	-	-
183.	Ranjani	29	III	Primi	T	-	-	-	23.2	-	110	-	-	-	-
184.	Gomaty	22	V	P	T	-	Oligo	-	21.05	-	53	-	-	-	-
185.	Sakntal	37	IV	$G_2P_1L_1$	T	-	-	-	23.06	-	94	-	-	-	-
186.	Jaya	24	IV	$G_3P_1L_1A_1$	T	-	-	-	21.78	-	99	-	-	-	-
187.	Vidhya	23	III	Primi	T	-	-	-	35.20	-	109	-	-	-	-
188.	Sumaty	27	IV	Primi	T	-	-	-	32.46	-	76	-	-	-	-
189.	Vidhya	25	IV	$G_2P_1L_0$	T	-	-	-	22.10	-	92	-	-	-	-
190.	Jayarai	28	V	$G_6P_2L_1A_3$	T	-	FMS	-	28.30	-	86	-	-	-	-
191.	Kavitha	28	II	Primi	T	-	PROM	-	25.96	-	112	-	-	-	-
192.	Hema	28	IV	Primi	T	-	-	-	26.47	-	110	-	-	-	-
193.	Jaya	24	IV	$G_2P_1L_1$	T	-	-	-	28.12	-	116	-	-	-	-
194.	Dhana	31	V	P	T	-	-	F	25.74	-	142	N	-	-	-
195.	Thanga	26	IV	$G_2P_1L_1$	T	-	-	-	27.23	-	142	N	-	-	-
196.	Kala	22	IV	Primi	T	-	PROM	-	25.47	-	120	-	-	-	-
197.	Sathya	20	III	Primi	T	-	Oligo	-	23.19	-	88	-	-	-	-
198.	Jaya	25	III	$G_2P_1L_1$	T	-	-	M	23.24	-	85	-	-	-	-
199.	Jine	27	III	$G_3P_2L_2$	T	-	-	-	26.15	-	101	-	-	-	-
200.	Chanda	23	III	$G_2P_1L_0$	T	ND	-	-	23.61	-	112	-	-	-	-
201	Sudha	22	III	P	T	-	-	-	25	-	120	-	-	-	-
202.	Jothi	28	V	$G_2P_1L_1$	T	-	-	-	26.87	-	98	-	-	-	-
203.	Rohini	26	V	$G_2P_1L_1$	T	Pr CS	-	-	20.81	-	110	-	-	-	-
204.	Jahana	27	II	Primi	PT	-	IUD	-	28.14	-	133	-	-	-	-
205.	Bhuvan	28	IV	$G_2P_1L_1$	T	-	-	-	26.33	-	117	-	-	-	-
206.	Rajes	28	IV	Primi	T	-	-	-	27.05	-	87	-	-	-	-
207.	Vijaya	27	IV	$G_2P_1L_1$	T	-	Oligo	-	17.07	-	106	-	-	-	-
208.	Nithya	22	IV	$G_3P_1L_1A_1$	T	-	-	-	27.94	-	94	-	-	-	-
209.	Shantha	26	III	Primi	T	-	-	F	24.12	-	130	-	-	-	-
210.	Kala	23	IV	$G_2P_1L_1$	T	Pr CS	Oligo	-	25	-	112	-	-	-	-
211.	Vennila	25	V	$G_2P_1L_1$	T	-	-	-	20.70	-	124	-	-	-	-
212.	Uma	34	II	$G_2P_1L_1$	T	-	PIH	-	27.12	-	106	-	-	-	-
213.	Sivgami	27	IV	Primi	T	-	PROM	-	24.28	-	108	-	-	-	-
214.	Stella	29	IV	$G_5P_2L_1A_1$	PT	Pr CS	-	-	30.30	-	80	-	-	-	-
215.	Dhana	23	III	$G_2P_1L_1$	PT	-	PROM	-	25.77	-	102	-	-	-	-
216.	Pomani	26	IV	$G_2P_1L_1$	T	Pr CS	-	-	26.75	-	130	-	-	-	-
217.	Meera	28	IV	$G_2P_1L_1$	T	Pr CS	-	-	27.20	-	92	-	-	-	-
218.	Aruna	41	III	$G_8P_1L_1A_6$	T	-	-	-	37.94	-	77	-	-	-	-
219.	Vaijaya	33	IV	$G_3P_1L_1A_1$	T	-	-	-	28.90	-	164	N	-	-	-
220.	Dhana	25	IV	$G_2P_1L_1$	T	-	-	-	24.17	-	140	N	-	-	
221.	Jaya	30	III	$G_2P_1L_1$	T	-	PIH+Oli	-	36.16	-	140	N	-	-	
222.	Sreeja	27	II	$G_2P_1L_1$	T	-	PROM	-	24.73	-	141	N	-	-	
223.	Vimala	27	II	$G_2P_1L_1$	PT	-	GDM	-	43.41	-	183	+	N	I	↑
224.	Thulasi	23	III	$G_2P_1L_1$	T	-	-	-	33.59	-	123	-	-	-	-
225.	Chitra	25	III	Primi	PT	-	PIH	-	25.91	-	116	-	-	-	-
226.	Nirmala	25	IV	G_4A_3	T	-	-	-	24	-	96	-	-	-	-
227.	Parmes	25	IV	Primi	T	-	-	-	18.15	-	109	-	-	-	-
228.	Kala	24	IV	$G_2P_1L_1$	T	Pr CS	-	-	24.1	-	104	-	-	-	-
229.	Nithya	22	IV	Primi	T	-	Oligo	-	21.6	-	108	-	-	-	-
230.	Deepa	24	IV	$G_2P_1L_1$	T	-	-	-	28.37	-	110	-	-	-	-
231.	Sreemat	26	III	$G_2P_1L_1$	PT	-	-	MF	39.6	-	107	-	-	-	-
232.	Bhuva	26	V	$G_4P_1L_1A_2$	T	-	-	-	25.10	-	107	-	-	-	-
233.	Kavitha	20	V	Primi	T	-	-	-	21.09	-	72	-	-	-	-
234.	Vinodh	28	II	$G_2P_1L_1$	T	Pr CS	-	-	21.09	-	121	-	-	-	-
235.	Thilaga	31	IV	$G_3P_1L_1A_1$	T	Pr CS	Oligo	-	23.80	-	140	N	-	-	-
236.	Vijaya	24	IV	G_2A_1	T	-	-	-	33.33	-	142	N	-	-	-
237.	Lavana	25	IV	$G_3P_1L_1A_1$	T	-	GDM	-	28.30	-	183	+	N	D	N
238.	Leena	31	III	$G_3P_1L_1A_1$	T	Pr CS	-	M	25.60	-	149	N	-	-	-
239.	Padma	23	IV	Primi	T	-	PIH	M	26.17	-	123	-	-	-	-
240.	Vijaya	25	III	Primi	T	-	PROM	-	29.68	-	97	-	-	-	-
241.	Sophia	29	III	Primi	T	-	PROM	F	25	-	115	-	-	-	-
242.	Vasanth	30	III	$G_3P_1L_1A_1$	T	-	-	-	23.17	-	91	-	-	-	-
243.	Abirami	26	III	Primi	T	-	PROM	-	22.15	-	91	-	-	-	-
244.	Bhuvan	29	IV	$G_2P_1L_0$	T	-	-	-	25.51	-	84		-	-	-
245.	Shanthi	29	III	Primi	T	-	-	-	28.51	-	106	-	-	-	-
246.	Juliet	38	III	$G_3P_1L_1A_1$	T	-	-	-	27.73	-	141	N	-	-	-
247.	Praveen	24	II	$G_2P_1L_1$	T	-	-	-	28.01	-	108	-	-	-	-

248.	Muthu	21	IV	$G_2P_1L_0$	T	Pr CS	-	-	26.56	-	123	-	-	-	-
249.	Basmat	30	IV	G_2A_1	T	-	PROM	-	25.31	-	120	-	-	-	-
250.	Radha	21	IV	Primi	T	Rh –	-	-	28.01	-	104	-	-	-	-
251.	Nithya	24	III	G_2A_1	PT	-	PTL	-	25.31	-	93	-	-	-	-
252.	Sathya	27	IV	$G_2P_1L_1$	T	Pr. CS	-	F	20.73	-	64	-	-	-	-
253.	Faridha	23	IV	Primi	T	-	-	-	26.49	-	75	-	-	-	-
254.	Uma	28	IV	Primi	T	Rh –-	PROM	-	24.21	-	85	-	-	-	-
255.	Devi	22	IV	Primi	T	-	-	-	23.43	-	142	N	-	-	-
256.	Sunitha	27	III	Primi	T	-	PIH	-	26.38	-	141	N	-	-	-
257.	Supriya	20	III	Primi	PT	-	PROM	-	17.72	-	81	-	-	-	-
258.	Jaya	21	IV	G_2A_1	T	-	-	-	23.25	-	92	-	-	-	-
259.	Subathr	23	IV	Primi	T	-	-	-	22.22	-	97	-	-	-	-
260.	Rani	22	IV	$G_3P_2L_1$	T	Pr. CS	-	-	19.53	-	105	-	-	-	-
261.	Rama	23	IV	Primi	T	-	PIH	M	24.21	-	106	-	-	-	-
262.	Tamil	22	IV	Primi	T	-	-	-	28.21	-	111	-	-	-	-
263.	Nirma	22	III	Primi	T	-	-	-	23.01	-	77	-	-	-	-
264.	Devi	25	IV	$G_2P_1L_1$	T	-	PIH	-	32.87	-	92	-	-	-	-
265.	Vasanta	25	IV	$G_3P_2L_0$	T	Pr. CS	Anen	-	26.56	-	142	N	-	-	-
266.	Saburni	29	V	$G_3P_2L_2$	T	-	-	-	26.78	-	146	N	-	-	-
267.	Priscilla	32	IV	Primi	T	-	-	-	26.89	-	108	-	-	-	-
268.	Jeenath	19	IV	Primi	PT	-	-	-	22	-	71	-	-	-	-
269.	Devi	22	III	Primi	PT	-	Breec.	-	29.77	-	106	-	-	-	-
270.	Jaya	21	IV	Primi	T	-	-	-	23	-	72	-	-	-	-
271.	Angala	24	III	Primi	T	-	-	-	22	-	108	-	-	-	-
272.	Hema	31	II	Primi	T	-	-	M	30.22	-	124	-	-	-	-
273.	Sheeba	25	III	$G_2P_1L_1$	T	-	-	-	26.33	-	95	-	-	-	-
274.	Vanitha	23	III	$G_2P_1L_1$	T	Pr.CS	-	-	26	-	89	-	-	-	-
275.	Naga	27	III	$G_3P_2L_1$	T	Pr. CS	-	-	25.77	-	121	-	-	-	-
276.	Amuda	20	IV	Primi	T	-	-	-	22.63	-	99	-	-	-	-
277.	Lavan	25	III	Primi	T	-	PIH	-	25.31	-	98	-	-	-	-
278.	Raja	30	IV	$G_2P_1L_1$	T	Pr. CS	-	-	26.56	-	76	-	-	-	-
279.	Maha	25	II	$G_2P_1L_1$	T	-	-	-	21.8	-	108	-	-	-	-
280.	Revathy	32	V	$G_2P_1L_1$	T	-	PROM	-	22.35	-	80	-	-	-	-
281.	Roopa	24	IV	Primi	T	-	-	-	22.22	-	88	-	-	-	-
282.	Latha	31	IV	$G_4P_1L_1A_2$	PT	-	PTL	-	33.3	-	120	-	-	-	-
283.	Sushela	30	III	Primi	T	-	-	-	23.4	-	92	-	-	-	-
284.	Vennila	30	III	$G_2P_1L_1$	T	-	-	-	24.88	-	123	-	-	-	-
285.	Muthu	36	V	$G_2P_1L_0$	T	Pr. CS	PIH	-	22.5	-	80	-	-	-	-
286.	Sumaty	28	III	G_3A_2	T	-	-	F	21.9	-	110	-	-	-	-
287.	Mohana	24	IV	$G_3P_2L_2$	T	-	-	-	23.16	-	108	-	-	-	-
288.	Mahes	32	III	Primi	T	-	-	M	28.5	-	132	-	-	-	-
289.	Sumaya	23	IV	Primi	T	Rh	-	-	21.3	-	108	-	-	-	-
290.	Vijaya	19	IV	Primi	T	-	-	-	23.5	-	108	-	-	-	-
291.	Sasi	27	V	$G_2P_1L_1$	T	-	-	-	31.01	-	109	-	-	-	-
292.	Vanaja	22	III	Primi	T	-	-	-	21.3	-	117	-	-	-	-
293.	Fasila	24	IV	$G_3P_2L_2$	T	-	-	-	23.93	-	116	-	-	-	-
294.	Uma	20	IV	Primi	T	-	-	-	21.36	-	117	-	-	-	-
295.	Priya	27	IV	$G_2P_1L_1$	T	-	-	-	29	-	98	-	-	-	-
296.	Kulanda	23	V	Primi	T	-	-	-	22.0	-	101	-	-	-	-
297.	Leema	30	III	$G_2P_1L_1$	T	-	-	-	28.20	-	128	-	-	-	-
298.	Priya	21	IV	Primi	T	-	-	-	26.01	-	101	-	-	-	-
299.	I	23	V	$G_2P_1L_1$	T	-	-	-	24	-	76	-	-	-	-
300.	Geetha	28	V	$G_2P_1L_1$	T	-	-	-	26.5	-	114	-	-	-	-
301.	Kavitha	23	V	Primi	T	-	-	-	23.87	-	61	-	-	-	-
302.	Swetha	25	V	$G_2P_1L_1$	T	Rh –	-	-	29.2	-	26	-	-	-	-
303.	Sumaty	32	IV	Primi	T	-	-	-	22.45	-	125	-	-	-	-
304.	Thilaga	27	IV	G_3A_2	T	-	Oligo	-	21.24	-	65	-	-	-	-
305.	Neelam	29	IV	$G_3P_2L_2$	T	-	-	-	27.07	-	106	-	-	-	-
306.	Vijaya	23	IV	$G_4P_1L_1A_2$	T	-	-	-	31.09	-	100	-	-	-	-
307.	Aruna	23	III	Primi	T	-	-	-	25.07	-	114	-	-	-	-
308.	Tamil	23	IV	$G_2P_1L_0$	T	-	-	-	20.31	-	134	-	-	-	-
309.	Saras	24	IV	Primi	T	Oligo	-	-	25.05	-	128	-	-	-	-
310.	Jaya	32	IV	$G_2P_1L_1$	T	-	-	-	24	-	109	-	-	-	-
311.	Deepa	25	III	Primi	T	-	Oligo	-	27.08	-	94	-	-	-	-
312.	Shantha	28	III	$G_5P_1L_1A_3$	T	-	-	-	23.05	-	107	-	-	-	-
313.	Kala	20	IV	Primi	T	-	-	-	23.05	-	87	-	-	-	-

314.	Shanm	21	III	Primi	PT	-	PIH	-	23.05	-	134	-	-	-	-
315.	Saroja	21	IV	Primi	T	-	PIH	-	25.03	-	90	-	-	-	-
316.	Aruna	23	III	Primi	T	-	-	-	25.04	-	114	-	-	-	-
317.	Hasina	23	IV	$G_2P_1L_1$	T	-	Bree	-	37.36	-	145	N	-	-	-
318.	Latha	26	IV	G_2A_1	T	-	Oligo	-	27.05	-	147	N	-	-	-
319.	Meera	29	III	G_3A_2	T	-	-	F	28	-	145	N	-	-	-
320.	Radha	27	IV	$G_3P_1L_1A_1$	T	Pr CS	-	-	25.09	-	162	+	N	D	N
321.	Padma	26	IV	G_2A_1	T	-	PIH	-	28.63	-	130	-	-	-	-
322.	Radhika	24	II	Primi	T	-	PROM	-	25.57	-	122	-	-	-	-
323.	Navane	29	IV	$G_3P_1L_1A_1$	T	-	-	-	23.45	-	84	-	-	-	-
324.	Kumdh	31	V	Primi	T	-	-	-	25.45	-	95	-	-	-	-
325.	Prema	32	IV	$G_3P_1L_1A_1$	T	-	-	-	25.28	-	100	-	-	-	-
326.	Gowri	24	IV	Primi	T	-	Hydr	-	17.77	-	86	-	-	-	-
327.	Maria	23	V	$G_4P_1L_1A_2$	T	NDAne	PTL	F	28.76	-	158	N	-	-	-
328.	Kales	22	IV	Primi	T	-	GDM	-	25	-	146	+	N	D	N
329.	Akila	28	IV	$G_2P_1L_1$	T	-	GDM	F	29.77	-	152	+	N	D	N
330.	Sridevi	24	III	G_2A_1	T	-	-	-	28.2	-	140	N	-	-	-
331.	Naga	24	IV	$G_2P_1L_1$	PT	Pr CS	PROM	-	29.32	-	101	-	-	-	-
332.	Bhuva	27	III	Primi	T	-	PROM	F	28.1	-	93	-	-	-	-
333.	Amuda	28	V	Primi	T	-	-	-	22.22	-	84	-	-	-	-
334.	Kavitha	27	V	$G_4P_1L_1A_2$	T	-	-	-	30.83	-	103	-	-	-	-
335.	Vijaya	23	V	Primi	T	-	-	-	20.48	-	75	-	-	-	-
336.	Yogam	29	IV	Primi	T	-	-	-	28.69	-	96	-	-	-	-
337.	Revathi	32	IV	$G_2P_1L_1$	T	-	-	M	24.6	-	89	-	-	-	-
338.	Mani	25	III	$G_3P_1L_1A_1$	T	-	Breec	-	21.92	-	128	-	-	-	-
339.	Gomaty	24	IV	Primi	T	-	-	-	27.98	-	117	-	-	-	-
340.	Revathy	22	IV	$G_2P_1L_1$	T	-	-	-	21.94	-	86	-	-	-	-
341.	Geetha	32	III	$G_3P_1L_1A_1$	T	Pr CS	-	-	28.57	-	116	-	-	-	-
342.	Nithya	25	II	G_2A_1	T	-	-	-	28.90	-	102	-	-	-	-
343.	Hariha	30	III	$G_3P_1L_1A_1$	T	-	-	-	29.00	-	149	N	-	-	-
344.	Anita	25	IV	Primi	T	-	-	-	25	-	144	N	-	-	-
345.	Rukman	40	IV	Primi	T	PIH	-	-	31.68	-	140	N	-	-	-
346.	Sabari	26	IV	G_4A_3	T	-	Se.PIH	-	30.91	-	113	-	-	-	-
347.	Jothi	26	IV	Primi	T	PIH	-	-	35.39	-	111	-	-	-	-
348.	Laksmi	32	V	$G_5P_1L_1A_3$	T	-	PIH	-	26.40	-	89	-	-	-	-
349.	Vijaya	29	III	$G_2P_1L_1$	T	-	-	-	31.74	-	121	-	-	-	-
350.	Visalatc	24	III	$G_2P_1L_1$	T	-	-	-	24.90	-	89	-	-	-	-
351.	Kalpana	25	III	$G_3P_1L_1A_1$	T	-	Anae	-	23.3	-	146	N	-	-	-
352.	Shanthi	24	III	$G_2P_1L_0A_0$	T	-	Oligo	-	24.9	-	140	N	-	-	-
353.	Akila	23	IV	Primi	T	-	-	-	23.66	-	88	-	-	-	-
354.	Kokila	24	IV	$G_2P_1L_1A_0$	T	Pr CS	-	-	25	-	86	-	-	-	-
355.	Nagajoti	21	IV	Primi	T	-	Rh –	-	18.77	-	97	-	-	-	-
356.	Then	20	III	Primi	T	-	PROM	-	24.46	-	102	-	-	-	-
357.	Sarojini	33	III	Primi	T	-	PIH	-	26.47	-	130	-	-	-	-
358.	Tharani	24	III	Primi	T	-	-	-	22.53	-	75	-	-	-	-
359.	Kokila	19	IV	Primi	T	-	-	-	25.51	-	102	-	-	-	-
360.	Yasoda	23	IV	Primi	T	-	-	-	24.2	-	124	-	-	-	-
361.	Krishna	27	IV	$G_2P_1L_1$	T	-	-	-	17.67	-	93	-	-	-	-
362.	Vijaya	24	IV	G_2A_1	T	-	-	-	36.37	-	112	-	-	-	-
363.	Munira	32	III	$G_4P_2L_2A_1$	T	-	-	-	25.49	-	58	-	-	-	-
364.	Devani	28	III	Primi	T	-	-	-	24.30	-	115	-	-	-	-
365.	Helen	26	III	Primi	T	-	Oilgo	-	23.33	-	81	-	-	-	-
366.	Vadivu	22	III	Primi	T	-	PIH	M	27.71	-	122	-	-	-	-
367.	Mohana	24	IV	$G_2P_1L_1$	T	-	-	-	16.42	-	90	-	-	-	-
368.	Deepa	24	IV	Primi	T	-	-	-	25	-	85	-	-	-	-
369.	Siri	26	III	Primi	T	-	-	-	23.1	-	116	-	-	-	-
370.	Thama	24	III	$G_2P_1L_0$	T	IUD	-	-	25	-	114	-	-	-	-
371.	Archna	19	IV	Primi	T	-	Breec	-	29.67	-	97	-	-	-	-
372.	Chandr	27	IV	$G_2P_1L_1$	T	-	-	-	26.08	-	81	-	-	-	-
373.	Radh	27	IV	$G_2P_1L_1$	T	-	PIH	-	20.46	-	83	-	-	-	-
374.	Esther	25	IV	$G_2P_1L_1$	T	-	-	-	26.07	-	99	-	-	-	-
375.	Sindhu	27	IV	$G_2P_1L_1$	T	-	-	-	22.23	-	112	-	-	-	-
376.	Laksmi	23	IV	$G_2P_1L_1$	T	-	-	-	23.17	-	108	-	-	-	-
377.	Yasoda	20	IV	Primi	T	-	-	-	24.47	-	116	-	-	-	-
378.	Rathina	22	V	$G_2P_1L_1$	T	-	-	-	20.08	-	108	-	-	-	-
379.	Mantha	25	IV	Primi	T	-	-	-	26.33	-	142	-	-	-	-

380.	Petchia	30	IV	$G_4P_1L_1A_2$	T	-	-	-	30.37	-	108	-	-	-	-
381.	Sasia	30	IV	$G_4P_1L_1A_2$	T	Anom	GDMPr	-	27	-	162	+	N	D	N
382.	Balam	23	IV	$G_2P_1L_0$	T	SB	GDMPI	-	20.8	-	148	+	N	D	N
383.	Chitra	24	IV	$G_3P_1L_1A_1$	T	Pr CS	-	-	23.4	-	101	-	-	-	-
384.	Maha	24	III	Primi	T	-	-	-	27.34	-	128	-	-	-	-
385.	Sabitha	22	III	Primi	T	-	-	-	27.01	-	116	-	-	-	-
386.	Amuda	28	III	$G_2P_1L_1$	T	Pr CS	-	-	25.7	-	94	-	-	-	-
387.	Alamu	29	IV	Primi	T	-	-	-	24.2	-	110	-	-	-	-
388.	Geetha	20	IV	Primi	T	-	-	M	33.5	-	81	-	-	-	-
389.	Niranja	22	III	Primi	T	-	-	-	36.7	-	92	-	-	-	-
390.	Shanm	30	III	$G_2P_1L_1$	T	-	Oligo	-	27.3	-	126	-	-	-	-
391.	Kumuta	31	III	$G_3P_1L_1A_1$	T	Pr CS	-	-	21.8	-	157	N	-	-	-
392.	Vijaya	30	III	Primi	T	-	-	-	21.4	-	113	-	-	-	-
393.	Jayanti	30	III	$G_4P_1L_1A_2$	T	-	-	-	34.9	-	109	-	-	-	-
394.	Malar	21	IV	Primi	T	-	-	-	27.3	-	96	-	-	-	-
395.	Selvi	28	III	Primi	T	-	-	-	28.6	-	87	-	-	-	-
396.	Angelin	29	III	Primi	T	-	-	-	27.3	-	97	-	-	-	-
397.	Nirmala	37	III	Primi	T	-	-	-	26	-	80	-	-	-	-
398.	Renuka	24	IV	Primi	T	-	PIH	M	22.1	-	68	-	-	-	-
399.	Fathima	24	IV	$G_6P_3L_2A_2$	T	-	-	-	30.2	-	95	-	-	-	-
400.	Santha	27	III	$G_2P_1L_1$	T	Pr CS	-	-	32.8	-	102	-	-	-	-
401.	Sugana	23	IV	$G_2P_1L_1$	T	-	-	-	25.3	-	111	-	-	-	-
402.	Priya	20	IV	G_2A_1	T	-	-	-	23.1	-	107	-	-	-	-
403.	Megala	23	IV	Primi	T	-	Oligo	-	26.3	-	85	-	-	-	-
404.	Padma	29	III	$G_2P_1L_1$	T	-	HIV +	-	31.2	-	134	-	-	-	-
405.	Surya	27	IV	$G_2P_1L_1$	T	-	Breec	-	24.8	-	133	-	-	-	-
406.	Parimal	23	III	Primi	PT	-	-	-	30.8	-	120	-	-	-	-
407.	Kalpana	23	IV	Primi	T	-	-	-	28.8	-	85	-	-	-	-
408.	Vijaya	25	IV	$G_3P_1L_0A_1$	T	Pr CS	-	-	26.6	-	106	-	-	-	-
409.	Savithiri	23	V	$G_2P_1L_1$	T	Pr CS	-	-	23.4	-	75	-	-	-	-
410.	Priya	26	III	$G_2P_1L_1$	T	-	-	-	29.2	-	122	-	-	-	-
411.	Jilidon	32	III	$G_2P_1L_1$	PT	-	PROM	-	36.8	-	164	N	-	-	-
412.	Radha	31	IV	Primi	T	-	PIH	F	27.4	-	142	N	-	-	-
413.	Jamuna	25	III	Primi	T	-	-	-	32.82	-	164	N	-	-	-
414.	Chitra	26	III	Primi	T	-	GDM	-	28.69	-	153	+	↑	I	↑
415.	Jothi	26	IV	$G_2P_1L_0$	T	ND	GDM	-	28.6	-	210	+	N	I	N
416.	Deepa	26	III	Primi	T	-	GDM	-	35.1	-	214	+	N	I	N
417.	Meena	32	III	$G_2P_1L_1$	T	4.2kg	GDM hy	MF	31.6	-	180	+	N	I	N
418.	Margat	26	V	G_2A_1	T	-	GDM	-	27.2	-	159	+	N	I	N
419.	Nalini	24	III	Primi	T	-	GDM	-	31.1	-	145	+	N	I	N
420.	Renu	37	II	$G_2P_1L_1$	T	Pr. CS	GDM hy	-	36.2	-	222	+	N	D	N
421.	Balmani	27	IV	$G_4P_1L_1A_2$	T	-	GDM	M	32.02	-	142	+	N	D	N
422.	Lakhmi	29	IV	$G_3P_1L_0A_1$	T	PIHIUD	GDM	M	26.9	-	140	+	N	D	N
423.	Prabha	25	IV	Primi	T	-	-	-	24.4	-	110	-	-	-	-
424.	Sudha	29	III	$G_3P_1L_1A_1$	T	-	PROM	-	33.3	-	111	-	-	-	-
425.	Anandi	24	IV	Primi	T	-	-	-	27.32	-	100	-	-	-	-
426.	Muthu	21	V	Primi	T	-	PROM	-	21.4	-	87	-	-	-	-
427.	Ponutai	25	IV	$G_2P_1L_1$	PT	-	-	-	23.4	-	100	-	-	-	-
428.	Sindu	19	IV	Primi	T	-	Hydra	-	24.4	-	74	-	-	-	-
429.	Revathy	26	IV	$G_2P_1L_0$	T	BOH	-	-	25.3	-	110	-	-	-	-
430.	Viola	24	III	P	T	-	-	F	26.62	-	108	-	-	-	-
431.	Priya	21	IV	P	T	-	PROM	F	24.1	-	87	-	-	-	-
432.	Kavitha	23	V	P	PT	-	-	-	23.0	-	90	-	-	-	-
433.	Prema	19	III	P	T	-	-	-	35.5	-	82	-	-	-	-
434.	Jaisee	31	III	P	T	-	-	-	28.42	-	82	-	-	-	-
435.	Mahes	22	V	Primi	T	-	-	-	24	-	135	-	-	-	-
436.	Girija	33	II	Primi	T	Epilep	-	-	24.4	-	120	-	-	-	-
437.	Nagama	26	V	$G_3P_1L_1A_1$	T	-	-	-	27.5	-	128	-	-	-	-
438.	Deepa	23	IV	$G_2P_1L_1$	T	PROM	-	F	25.32	-	120	-	-	-	-
439.	Saras	21	III	Primi	T	-	-	-	27.02	-	128	-	-	-	-
440.	Shobna	27	IV	$G_2P_1L_1$	T	-	-	-	27.02	-	114	-	-	-	-
441.	Usha	35	III	$G_5P_1L_1A_3$	T	-	-	-	24	-	108	-	-	-	-
442.	Jihan	23	III	G_2A_1	PT	PIH	-	-	28	-	100	-	-	-	-
443.	Gowri	20	IV	$G_2P_1L_1$	T	-	Oligo	-	29.3	-	104	-	-	-	-
444.	Firoja	19	IV	Primi	T	-	-	-	22.4	-	86	-	-	-	-
445.	Kowsik	27	III	Primi	T	-	-	-	24.6	-	123	-	-	-	-

446.	Joyjona	26	III	G_2A_1	T	-	-	-	21.9	-	80	-	-	-	-
447.	Deepa	21	IV	G_3A_2	T	-	-	-	28.2	-	75	-	-	-	-
448.	Alafia	35	IV	$G_3P_2L_2$	T	PIH	PIH	M	26.4	-	135	+	-	-	-
449.	Sreeba	24	II	$G_3P_1L_1A_1$	T	-	-	-	28.4	-	85	-	-	-	-
450.	Kala	22	IV	Primi	T	-	-	-	21.6	-	113	-	-	-	-
451.	Freeda	34	III	$G_3P_1L_1A_1$	T	-	-	-	21	-	124	-	-	-	-
452.	Jaya	21	III	Primi	T	-	-	-	22.6	-	84	-	-	-	-
453.	Nabisha	37	IV	Primi	PT	-	Oligo	-	26.4	-	82	-	-	-	-
454.	Leela	27	III	Primi	T	-	-	-	27.2	-	122	-	-	-	-
455.	Julie	33	IV	$G_5P_1L_1A_3$	T	-	PIH	-	31.5	-	131	-	-	-	-
456.	Kiritika	21	V	Primi	T	-	-	-	23.2	-	77	-	-	-	-
457.	Kalpana	26	III	Primi	T	-	Oligo	-	25.4	-	118	-	-	-	-
458.	Kasturi	25	III	Primi	T	Rh -	-	-	31.3	-	108	-	-	-	-
459.	Jothi	23	IV	Primi	T	-	PROM	-	20.8	-	110	-	-	-	-
460.	Megala	27	III	Primi	T	-	-	-	27	-	114	-	-	-	-
461.	Janaki	28	IV	$G_3P_1L_1A_1$	T	-	-	-	22.4	-	80	-	-	-	-
462.	Aruselvi	27	V	G_2A_1	PT	-	-	-	19.5	-	90	-	-	-	-
463.	Bagya	31	IV	$G_2P_1L_1$	T	-	-	-	22.2	-	93	-	-	-	-
464.	Jasmin	26	IV	$G_3P_1L_1A_1$	T	-	-	-	26.1	-	106	-	-	-	-
465.	Baby	25	III	$G_2P_1L_1$	T	-	-	-	23.6	-	117	-	-	-	-
466.	Geetha	32	IV	$G_2P_1L_1$	T	Pr CS	-	-	22.8	-	156	-	-	-	-
467.	Vasant	30	III	Primi	T	-	Oligo	-	20.9	-	94	-	-	-	-
468.	Pushpa	29	V	$G_2P_1L_1$	T	Pr CS	-	-	25	-	100	-	-	-	-
469.	Sumaya	28	IV	$G_2P_1L_1$	T	-	-	-	25.6	-	74	-	-	-	-
470.	Sameen	26	III	Primi	T	-	-	F	22.7	-	144	N	-	-	-
471.	Saras	30	V	$G_3P_2L_1$	T	IUD	-	-	32.5	-	140	N	-	-	-
472.	Valar	19	IV	Primi	T	-	Oligo	-	33	-	111	+	-	-	-
473.	Padma	25	V	Primi	T	Polio	-	-	27.6	-	83	-	-	-	-
474.	Mahesh	25	IV	$G_4P_2L_1A_1$	T	PrCSIUD	-	-	26		118	-			
475.	Selva	23	II	Primi	T	-	-	-	24.8	-	113	-	-	-	-
476.	Rathi	26	IV	Primi	PT	PPROM	Breec	-	26.7	-	113	-	-	-	-
477.	Radha	23	IV	Primi	T	-	-	-	24.7	-	82	-	-	-	-
478.	Pushpa	32	III	$G_2P_1L_1$	T	-	-	-	25.9	-	115	-	-	-	-
479.	Asha	30	III	$G_2P_1L_1$	T	-	PROM	-	22.4	-	131	-	-	-	-
480.	Lalitha	24	III	$G_2P_1L_1$	T	-	OliPIH	-	29.2	-	107	-	-	-	-
481.	Iyamuth	23	IV	Primi	T	-	-	-	27.3	-	70	-	-	-	-
482.	Asraf	25	V	$G_2P_1L_1$	T	Pr CS	-	-	31.6	-	151	N	-	-	-
483.	Selva	26	III	Primi	T	-	-	F	28.5	-	178	N	-	-	-
484.	Prabha	28	IV	Primi	T	-	-	-	23.4	-	133	-	-	-	-
485	Cyron	32	III	$G_2P_1L_1$	T	-	-	-	35.5	-	123	-	-	-	-
486.	Naga	28	II	$G_1P_1L_1A_1$	T		-	-	27.7	-	122	-	-	-	-
487.	Jaya	22	III	Primi	T	-	-	-	25	-	118	-	-	-	-
488.	Anitha	21	IV	Primi	T	-	Oligo	-	22.6	-	108	-	-	-	-
489.	Prema	25	IV	Primi	T	-	-	M	19.5	-	147	N	-	-	-
490.	Sudha	23	IV	G_2A_1	T	-	Rh –	-	23.4	-	150	N	-	-	-
491.	Tamil	28	IV	$G_2P_1L_1$	T	-	-	-	26	-	77	-	-	-	-
492.	Sridevi	24	IV	$G_2P_1L_1$	T	-	-	-	28.3		100	-	-	-	-
493.	Lilly	20	IV	Primi	T	-	-	-	26	-	108	-	-	-	-
494.	Jaba	21	V	Primi	T	-	-	-	27	-	110	-	-	-	-
495.	Kalpana	22	IV	Primi	T	-	-	-	22.4	-	108	-	-	-	-
496.	Rahda	23	IV	Primi	T	-	-	-	24	-	106	-	-	-	-
497.	Ananta	29	IV	G_2A_1	T	-	-	-	22	-	142	N	-	-	-
498.	Vasanta	27	III	$G_2P_1L_1$	T	-	-	-	21.2	-	112	-	-	-	-
499.	Mahes	19	IV	Primi	T	-	-	-	23	-	108	-	-	-	-
500.	Vidhya	23	III	Primi	T	-	GDM Oli	F	26	-	148	+	N	D	-
501.	Mani	29	III	$G_2P_1L_1$	T	-	-	-	23.2	-	112	-	-	-	-
502	Prema	33	IV	$G_2P_1L_1$	T	-	-	-	24.1	-	106	-	-	-	-

Lost for followup

503.	Kamala	25	III	$G_2P_1L_1$	T	-	-	-	23.6	-	117	-
504.	Vimala	32	IV	$G_2P_1L_1$	T	Pr CS	-	-	22.8	-	156	-
505.	Vasant	30	III	Primi	T	-	-	-	20.9	-	94	-
506.	Rani	29	V	$G_2P_1L_1$	T	Pr CS	-	-	25	-	100	-
507.	Jeni	28	IV	$G_2P_1L_1$	T	-	-	-	25.6	-	74	-

508.	**Sheeba**	**26**	**III**	**Primi**	**T**	**-**	**-**	**F**	**22.7**	**-**	**144**	**N**
509.	**Chitra**	**21**	**IV**	**G_3A_2**	**T**	**-**	**-**	**-**	**28.2**	**-**	**75**	**-**
510.	**Vimala**	**35**	**IV**	**$G_3P_2L_2$**	**T**	**PIH**	**-**	**M**	**26.4**	**-**	**135**	**+**
511.	**Kala**	**24**	**II**	**$G_3P_1L_1A_1$**	**T**	**-**	**-**	**-**	**28.4**	**-**	**85**	**-**
512.	**Selvi**	**22**	**IV**	**Primi**	**T**	**-**	**-**	**-**	**21.6**	**-**	**113**	**-**
513.	**Rani**	**34**	**III**	**$G_3P_1L_1A_1$**	**T**	**-**	**-**	**-**	**21**	**-**	**124**	**-**
514.	**Vijaya**	**21**	**III**	**Primi**	**T**	**-**	**-**	**-**	**22.6**	**-**	**84**	**-**
515.	**Mani**	**37**	**IV**	**Primi**	**PT**	**-**	**-**	**-**	**26.4**	**-**	**82**	**-**
516.	**Neela**	**27**	**III**	**Primi**	**T**	**-**	**-**	**-**	**27.2**	**-**	**122**	**-**
517.	**Jenny**	**33**	**IV**	**$G_5P_1L_1A_3$**	**T**	**-**	**-**	**-**	**31.5**	**-**	**131**	**-**
518.	**Kavitha**	**21**	**V**	**Primi**	**T**	**-**	**-**	**-**	**23.2**	**-**	**77**	**-**
519.	**Kalpana**	**26**	**III**	**Primi**	**T**	**-**	**-**	**-**	**25.4**	**-**	**118**	**-**
520.	**Revathy**	**25**	**III**	**Primi**	**T**	**Rh -**	**-**	**-**	**31.3**	**-**	**108**	**-**
521.	**Jothi**	**23**	**IV**	**Primi**	**T**	**-**	**-**	**-**	**20.8**	**-**	**110**	**-**
522.	**Megala**	**27**	**III**	**Primi**	**T**	**-**	**-**	**-**	**27**	**-**	**114**	**-**
523.	**Janaki**	**28**	**IV**	**$G_3P_1L_1A_1$**	**T**	**-**	**-**	**-**	**22.4**	**-**	**80**	**-**
524.	**Selvi**	**27**	**V**	**G_2A_1**	**PT**	**-**	**-**	**-**	**19.5**	**-**	**90**	**-**

Key words:

SES – Socio economic status
IUGR - Intra uterine growth restriction
T – Term
EMS - Emergency caeserean section
PT - Preterm
ELS - Elective caeserean section
Oligo – Oligohydramnios
N - Normal
Hydra - Hydramnios
D - Diet
GDM - Gestational diabetes mellitus
I - Insulin
Pr. CS - Previous caeserean section
V - Vaginal delivery
BOH - Bad obstetrics history
V-V - Vaccum
PIH - Pregnancy induced hypertension
ATF - Axis traction forceps
PROM- Pre labour rupture of membranes
I - Induced
F - Father
S - Spontaneous
M - Mother
HB - Hyperbilirubinaemia
GCT - Glucose challenge test
HG - Hypoglycaemia
GTT - Glucose tolerance test
RDS - Respiratory distress syndrome
TTN - Transient tachypnoea of newborn
Asphy. - Birth Asphyxia
LGA - Large for gestational age
SGA - Small for gestational age
GA - Gestational age
BMI - Bodymass index
Anen. - Anencephaly
HC - Hydrocephaly
ND - Neonatal death
SB - Still birth
Anom – Anomalous baby
PTL - Preterm labour